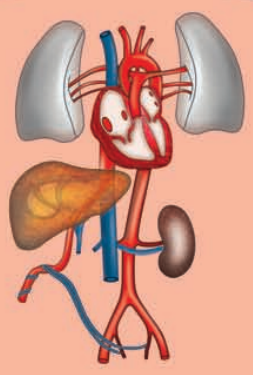
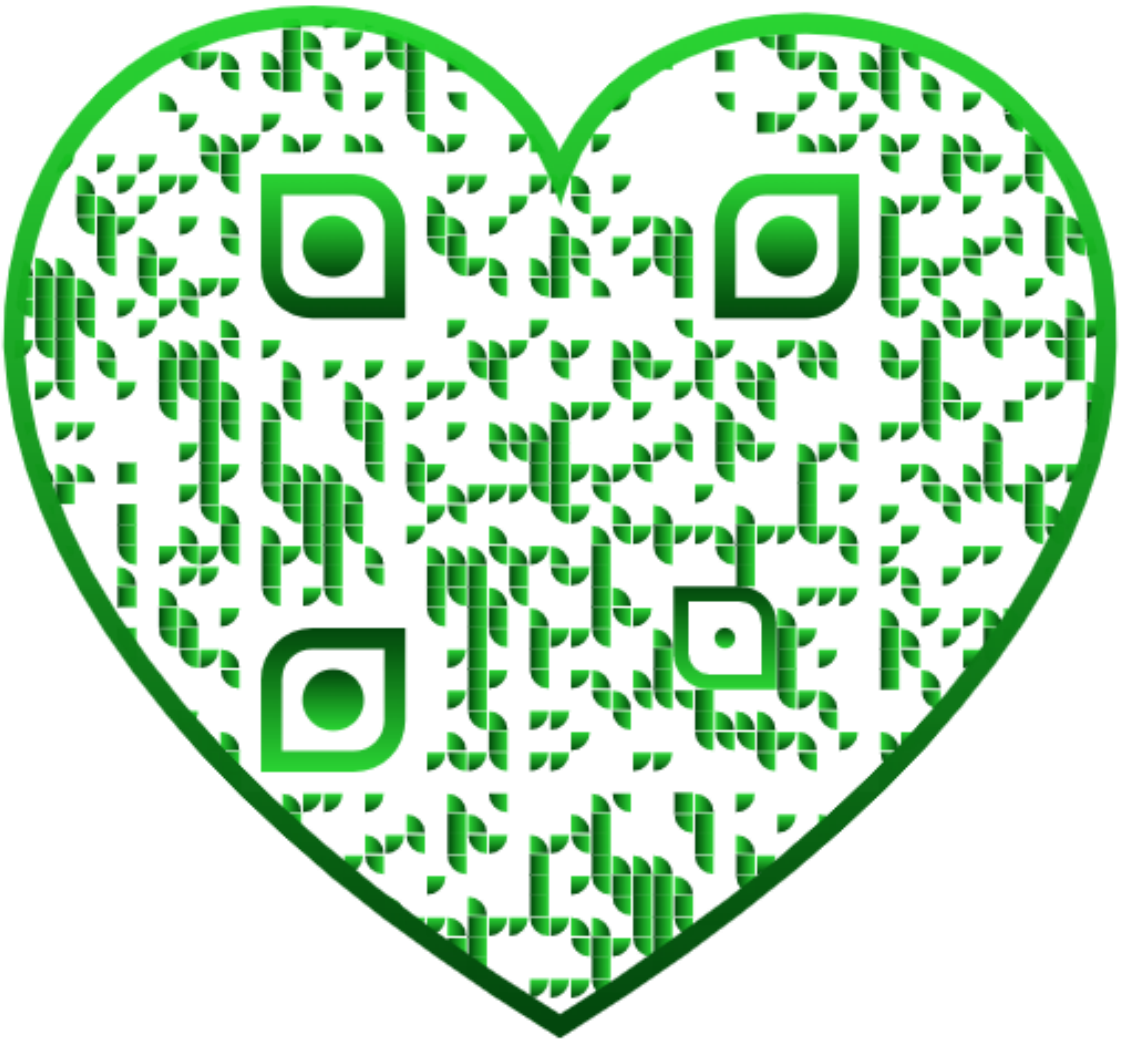
Section 8

Cardiovascular System

89. Introduction to Cardiovascular System 519
90. Properties of Cardiac Muscle 525
91. Cardiac Cycle 533
92. Heart Sounds 544
93. Cardiac Murmur 549
94. Electrocardiogram (ECG) 551
95. Vector 558
96. Arrhythmia 562
97. Effect of Changes in Electrolyte Concentration on Heart 570
98. Cardiac Output 572
99. Heart-lung Preparation 582
100. Cardiac Function Curves 584
101. Heart Rate 587
102. Hemodynamics 595
103. Arterial Blood Pressure 602
104. Venous Pressure 617
105. Capillary Pressure 620





106. Arterial Pulse 622
107. Venous Pulse 627
108. Coronary Circulation 629
109. Cerebral Circulation 634
110. Splanchnic Circulation 638
111. Capillary Circulation 640
112. Circulation through Skeletal Muscle 644
113. Cutaneous Circulation 646
114. Fetal Circulation and Respiration 648
115. Hemorrhage 651
116. Circulatory Shock and Heart Failure 654
117. Cardiovascular Adjustments during Exercise 664

Introduction to Cardiovascular System

Chapter 89

- CARDIOVASCULAR SYSTEM
- HEART
  - RIGHT SIDE
  - LEFT SIDE
  - SEPTA
  - LAYERS OF THE WALL
  - PERICARDIUM
  - MYOCARDIUM
  - ENDOCARDIUM
  - VALVES
- ACTIONS OF THE HEART
  - CHRONOTROPIC ACTION
  - INOTROPIC ACTION
  - DROMOTROPIC ACTION
  - BATHMOTROPIC ACTION
- BLOOD VESSELS
  - ARTERIAL SYSTEM
  - VENOUS SYSTEM
  - COMPLICATIONS IN BLOOD VESSELS
- DIVISIONS OF CIRCULATION
  - SYSTEMIC CIRCULATION
  - PULMONARY CIRCULATION

CARDIOVASCULAR SYSTEM

Cardiovascular system includes **heart** and **blood vessels.** Heart pumps blood into the blood vessels. Blood vessels circulate the blood throughout the body. Blood transports nutrients and oxygen to the tissues and removes carbon dioxide and waste products from the tissues.

HEART

Heart is a muscular organ that pumps blood throughout the circulatory system. It is situated in between two lungs in the mediastinum. It is made up of four chambers, two atria and two ventricles. The musculature of ventricles is thicker than that of atria. Force of contraction of heart depends upon the muscles.

RIGHT SIDE OF THE HEART

Right side of the heart has two chambers, **right atrium** and **right ventricle.** Right atrium is a thin walled and low pressure chamber. It has got the pacemaker known as sinoatrial node that produces cardiac impulses and atrioventricular node that conducts the impulses to the ventricles.

Right atrium receives venous (deoxygenated) blood via two large veins:

1. **Superior vena cava** that returns venous blood from the head, neck and upper limbs

2. **Inferior vena cava** that returns venous blood from lower parts of the body (Fig. 89.1).

Right atrium communicates with right ventricle through tricuspid valve. Wall of right ventricle is thick. Venous blood from the right atrium enters the right ventricle through this valve.

From the right ventricle, pulmonary artery arises. It carries the venous blood from right ventricle to lungs. In the lungs, the deoxygenated blood is oxygenated.

■ LEFT SIDE OF THE HEART

Left side of the heart has two chambers, **left atrium** and **left ventricle.** Left atrium is a thin walled and low pressure chamber. It receives oxygenated blood from the lungs through pulmonary veins. This is the only exception in the body, where an artery carries venous blood and vein carries the arterial blood.

Blood from left atrium enters the left ventricle through mitral valve (bicuspid valve). Wall of the left ventricle is very thick. Left ventricle pumps the arterial blood to different parts of the body through **systemic aorta.**

■ SEPTA OF THE HEART

Right and left atria are separated from one another by a fibrous septum called **interatrial septum.** Right and left ventricles are separated from one another by **interventricular septum.** The upper part of this septum is a membranous structure, whereas the lower part of it is muscular in nature.

■ LAYERS OF WALL OF THE HEART

Heart is made up of three layers of tissues:

1. Outer pericardium
2. Middle myocardium
3. Inner endocardium.

■ PERICARDIUM

Pericardium is the outer covering of the heart. It is made up of two layers:

i. Outer parietal pericardium
ii. Inner visceral pericardium.

The space between the two layers is called **pericardial cavity** or **pericardial space** and it contains a thin film of fluid.

i. *Outer Parietal Pericardium*

Parietal pericardium forms a strong protective sac for the heart. It helps also to anchor the heart within the mediastinum.

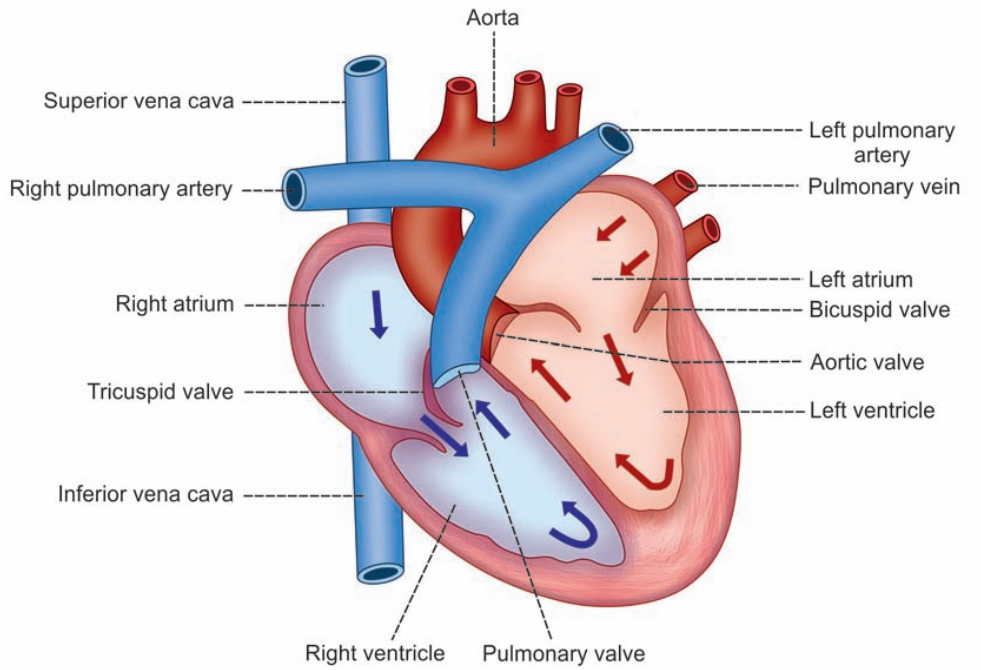


FIGURE 89.1: Section of the heart

Parietal pericardium is made up two layers:

a. Outer fibrous layer
b. Inner serous layer.

Fibrous layer

Fibrous layer of the parietal pericardium is formed by thick fibrous connective tissue. It is attached to the diaphragm and it is continuous with tunica adventitia (outer wall) of the blood vessels, entering and leaving the heart. It is attached with diaphragm below. Because of the fibrous nature, it protects the heart from over stretching.

Serous layer

Serous layer is formed by mesothelium, together with a small amount of connective tissue. Mesothelium contains squamous epithelial cells which secrete a small amount of fluid, which lines the pericardial space. This fluid prevents friction and allows free movement of heart within pericardium, when it contracts and relaxes. The total volume of this fluid is only about 25 to 35 mL.

ii. *Inner Visceral Pericardium*

Inner visceral pericardium lines the surface of myocardium. It is made up of flattened epithelial cells. This layer is also known as **epicardium.**

■ MYOCARDIUM

Myocardium is the middle layer of wall of the heart and it is formed by cardiac muscle fibers or cardiac myocytes. Myocardium forms the bulk of the heart and it is responsible for pumping action of the heart. Unlike skeletal muscle fibers, the cardiac muscle fibers are involuntary in nature.

Refer Chapter 28 for features of cardiac muscles.

Myocardium has three types of muscle fibers:

i. Muscle fibers which form contractile unit of heart
ii. Muscle fibers which form pacemaker
iii. Muscle fibers which form conductive system.

i. *Muscle Fibers which Form Contractile Unit of Heart*

These cardiac muscle fibers are striated and resemble the skeletal muscle fibers in structure. Cardiac muscle fiber is bound by **sarcolemma.** It has a centrally placed nucleus. **Myofibrils** are embedded in the sarcoplasm. **Sarcomere** of the cardiac muscle has all the contractile proteins, namely actin, myosin, troponin and tropomyosin. **Sarcotubular system** in cardiac muscle is similar to that of skeletal muscle.

Important difference between skeletal muscle and cardiac muscle is that the cardiac muscle fiber is branched and the skeletal muscle is not branched.

Intercalated disk

Intercalated disk is a tough double membranous structure, situated at the junction between the branches of neighboring cardiac muscle fibers. It is formed by the fusion of the membrane of the cardiac muscle branches (Fig. 89.2).

Intercalated disks form **adherens junctions,** which play an important role in the contraction of cardiac muscle as a single unit (Chapter 2).

Syncytium

Syncytium means tissue with cytoplasmic continuity between adjacent cells. However, cardiac muscle is like a **physiological syncytium,** since there is no continuity of the cytoplasm and the muscle fibers are separated from each other by cell membrane. At the sides, the membranes of the adjacent muscle fibers fuse together to form **gap junctions.** Gap junction is permeable to ions and it facilitates the rapid conduction of action potential from one fiber to another. Because of this, all the cardiac muscle fibers act like a single unit, which is referred as syncytium.

Syncytium in human heart has two portions, syncytium of atria and the syncytium of ventricles. Both the portions of syncytium are connected by a thick non-conducting fibrous ring called the **atrioventricular ring.**

ii. *Muscle Fibers which Form the Pacemaker*

Some of the muscle fibers of heart are modified into a specialized structure known as pacemaker. These muscle fibers forming the pacemaker have less striation.

Pacemaker

Pacemaker is structure in the heart that generates the impulses for heart beat. It is formed by **pacemaker cells**

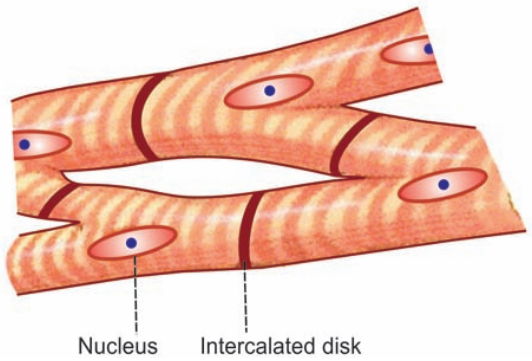


FIGURE 89.2: Cardiac muscle fibers

called **P cells.** Sinoatrial (SA) node forms the pacemaker in human heart. Details of pacemaker are given in next chapter.

iii. *Muscle Fibers which Form Conductive System*

Conductive system of the heart is formed by modified cardiac muscle fibers. Impulses from SA node are transmitted to the atria directly. However, the impulses are transmitted to ventricles through various components of conducting system, which are explained in the next chapter.

■ ENDOCARDIUM

Endocardium is the inner most layer of heart wall. It is a thin, smooth and glistening membrane. It is formed by a single layer of endothelial cells, lining the inner surface of the heart. Endocardium continues as endothelium of the blood vessels.

■ VALVES OF THE HEART

There are four valves in human heart. Two valves are in between atria and the ventricles called atrioventricular valves. Other two are the semilunar valves, placed at the opening of blood vessels arising from ventricles, namely systemic aorta and pulmonary artery. Valves of the heart permit the flow of blood through heart in only one direction.

Atrioventricular Valves

Left atrioventricular valve is otherwise known as **mitral valve** or **bicuspid valve.** It is formed by two valvular **cusps** or flaps (Fig. 89.3). Right atrioventricular valve is known as **tricuspid valve** and it is formed by three cusps.

Brim of the atrioventricular valves is attached to atrioventricular ring, which is the fibrous connection between the atria and ventricles. Cusps of the valves are attached to **papillary muscles** by means of **chordae tendineae.** Papillary muscles arise from inner surface of the ventricles. Papillary muscles play an important role in closure of the cusps and in preventing the back flow of blood from ventricle to atria during ventricular contraction.

Atrioventricular valves open only towards ventricles and prevent the backflow of blood into atria.

Semilunar Valves

Semilunar valves are present at the openings of systemic aorta and pulmonary artery and are known as **aortic valve** and **pulmonary valve** respectively. Because of the half moon shape, these two valves are called semilunar valves. Semilunar valves are made up of three flaps.

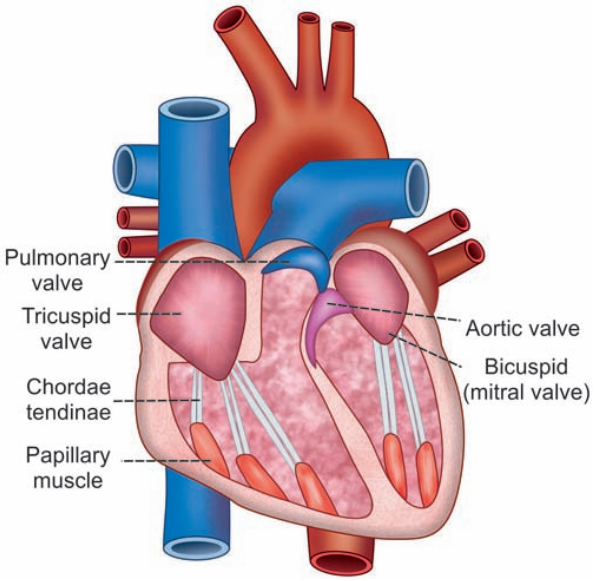


FIGURE 89.3: Valves of the heart

Semilular valves open only towards the aorta and pulmonary artery and prevent the backflow of blood into the ventricles.

■ ACTIONS OF THE HEART

Actions of the heart are classified into four types:

1. Chronotropic action
2. Inotropic action
3. Dromotropic action
4. Bathmotropic action.

■ CHRONOTROPIC ACTION

Chronotropic action is the frequency of heartbeat or heart rate. It is of two types:

i. **Tachycardia** or increase in heart rate
ii. **Bradycardia** or decrease in heart rate.

■ INOTROPIC ACTION

Force of contraction of heart is called inotropic action. It is of two types:

i. Positive inotropic action or increase in the force of contraction
ii. Negative inotropic action or decrease in the force of contraction.

■ DROMOTROPIC ACTION

Dromotropic action is the conduction of impulse through heart. It is of two types:

i. Positive dromotropic action or increase in the velocity of conduction
ii. Negative dromotropic action or decrease in the velocity of conduction.

■ BATHMOTROPIC ACTION

Bathmotropic action is the excitability of cardiac muscle. It is also of two types:

i. Positive bathmotropic action or increase in the excitability of cardiac muscle
ii. Negative bathmotropic action or decrease in the excitability of cardiac muscle.

Regulation of Actions of Heart

All the actions of heart are continuously regulated. It is essential for the heart to cope up with the needs of the body. All the actions are altered by stimulation of nerves supplying the heart or some hormones or hormonal substances secreted in the body.

■ BLOOD VESSELS

Vessels of circulatory system are the aorta, arteries, arterioles, capillaries, venules, veins and venae cavae. Structural differences between different blood vessels are given in Table 89.1.

■ ARTERIAL SYSTEM

Arterial system comprises the aorta, arteries and arterioles. Walls of the aorta and arteries are formed by three layers:

1. Outer **tunica adventitia,** which is made up of connective tissue layer. It is the continuation of fibrous layer of parietal pericardium.
2. Middle **tunica media,** which is formed by smooth muscles
3. Inner **tunica intima,** which is made up of endothelium. It is the continuation of endocardium.

Aorta, arteries and arterioles have two laminae of elastic tissues:

i. **External elastic lamina** between tunica adventitia and tunica media
ii. **Internal elastic lamina** between tunica media and tunica intima.

Aorta and arteries have more elastic tissues and the arterioles have more smooth muscles.

Arterial branches become narrower and their walls become thinner while reaching the periphery. Aorta has got the maximum diameter of about 25 mm. Diameter of the arteries is gradually decreased and at the end arteries, it is about 4 mm. It further decreases to 30 µ in the arterioles and ends up with 10 µ in the terminal arterioles. **Resistance** (peripheral resistance) is offered to blood flow in the arterioles and so these vessels are called **resistant vessels.**

Arterioles are continued as capillaries, which are small, thin walled vessels having a diameter of about 5 to 8 µ. Capillaries are functionally very important because, the exchange of materials between the blood and the tissues occurs through these vessels.

■ VENOUS SYSTEM

From the capillaries, venous system starts and it includes venules, veins and venae cavae. Capillaries end in venules, which are the smaller vessels with thin muscular wall than the arterioles. Diameter of the venules is about 20 µ. At a time, a large quantity of blood is held in venules and hence the venules are called **capacitance vessels.** Venules are continued as veins, which have the diameter of 5 mm. Veins form superior and inferior venae cavae, which have a diameter of about 30 mm.

TABLE 89.1: Structural and dimensional differences between different blood vessel walls

Blood vessel	Diameter	Thickness of the wall	Elastic tissue	Smooth muscle fibers	Fibrous tissue
Aorta	25 mm	2 mm	More	Less	More
Artery	4 mm	1 mm	More	More	Moderate
Arteriole	30 µ	6 µ	Moderate	More	Moderate
Terminal arteriole	10 µ	2 µ	Less	More	Moderate
Capillary	8 µ	0.5 µ	Absent	Absent	Moderate
Venule	20 µ	1 µ	Absent	Absent	Less
Vein	5 mm	0.5 mm	Less	More	Moderate
Vena cava	30 mm	1.5 mm	Less	More	More

Walls of the veins and venae cavae are made up of inner endothelium, elastic tissues, smooth muscles and outer connective tissue layer. In the veins and venae cavae, the elastic tissue is less but the smooth muscle fibers are more.

■ COMPLICATIONS IN BLOOD VESSELS

Aorta and Arteries

Arterial blood vessels are highly susceptible for arteriosclerosis and atherosclerosis. **Arteriosclerosis** is the disease of the arteries, associated with hardening, thickening and loss of elasticity in the wall of the vessels. **Atherosclerosis** is the disease marked by the narrowing of lumen of arterial vessel due to deposition of cholesterol.

Arterioles

When the tone of the smooth muscles in the arterioles increases, hypertension occurs.

Capillaries

Permeability of the capillary membrane may increase resulting in shock or edema due to leakage of fluid, proteins and other substances from blood.

Veins

Inflammation of the wall of veins leads to the formation of intravascular clot called **thrombosis.** The clot gets dislodged, as **thrombus.** The thrombus travels through blood and causes **embolism.** Embolism obstructs the blood flow to vital organs such as brain, heart and lungs, leading to many complications.

■ DIVISIONS OF CIRCULATION

Blood flows through two divisions of circulatory system:

1. Systemic circulation
2. Pulmonary circulation.

■ SYSTEMIC CIRCULATION

Systemic circulation is otherwise known as **greater circulation** (Fig. 89.4). Blood pumped from left ventricle passes through a series of blood vessels, arterial system

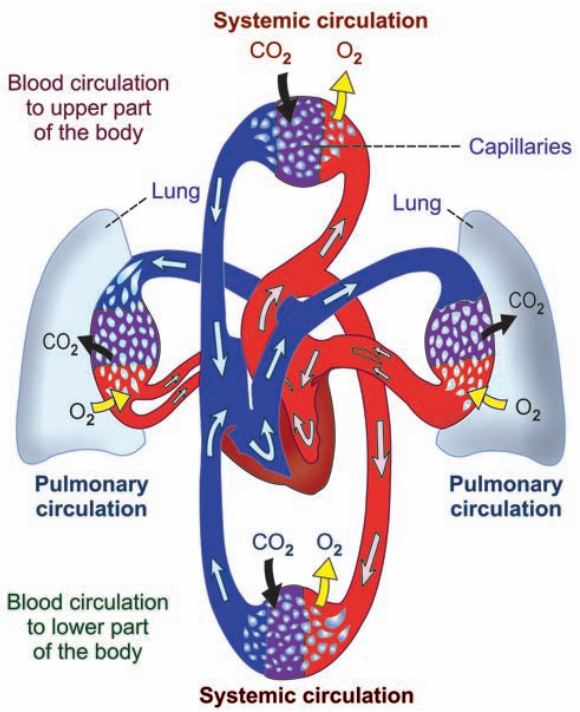


FIGURE 89.4: Systemic and pulmonary circulation

and reaches the tissues. Exchange of various substances between blood and the tissues occurs at the capillaries.

After exchange of materials, blood enters the venous system and returns to right atrium of the heart. From right atrium, blood enters the right ventricle.

Thus, through systemic circulation, oxygenated blood is supplied from heart to the tissues and venous blood returns to the heart from tissues.

■ PULMONARY CIRCULATION

Pulmonary circulation is otherwise called **lesser circulation.** Blood is pumped from right ventricle to lungs through pulmonary artery. Exchange of gases occurs between blood and alveoli of the lungs at pulmonary capillaries. Oxygenated blood returns to left atrium through the pulmonary veins.

Thus, left side of the heart contains oxygenated or arterial blood and the right side of the heart contains deoxygenated or venous blood.

Properties of Cardiac Muscle

Chapter 90

- EXCITABILITY
  - DEFINITION
  - ELECTRICAL POTENTIALS IN CARDIAC MUSCLE
  - IONIC BASIS OF ACTION POTENTIAL
  - SPREAD OF ACTION POTENTIAL THROUGH CARDIAC MUSCLE
- RHYTHMICITY
  - DEFINITION
  - PACEMAKER
  - ELECTRICAL POTENTIAL IN SINOATRIAL NODE
- CONDUCTIVITY
  - CONDUCTIVE SYSTEM IN HUMAN HEART
  - VELOCITY OF IMPULSES AT DIFFERENT PARTS OF CONDUCTIVE SYSTEM
- CONTRACTILITY
  - ALL-OR-NONE LAW
  - STAIRCASE PHENOMENON
  - SUMMATION OF SUBLIMINAL STIMULI
  - REFRACTORY PERIOD

EXCITABILITY

DEFINITION

Excitability is defined as the ability of a living tissue to give response to a stimulus. In all the tissues, initial response to a stimulus is electrical activity in the form of action potential. It is followed by mechanical activity in the form of contraction, secretion, etc.

ELECTRICAL POTENTIALS IN CARDIAC MUSCLE

Refer Chapter 31 for basics of electrical potentials in the muscle.

Resting Membrane Potential

Resting membrane potential in:

Single cardiac muscle fiber	:	– 85 to – 95 mV
Sinoatrial (SA) node	:	– 55 to – 60 mV
Purkinje fibers	:	– 90 to – 100 mV.

Action Potential

Action potential in cardiac muscle is different from that of other tissues such as skeletal muscle, smooth muscle and nervous tissue. Duration of the action potential in cardiac muscle is 250 to 350 msec (0.25 to 0.35 sec).

Phases of action potential

Action potential in a single cardiac muscle fiber occurs in four phases:

1. Initial depolarization
2. Initial repolarization
3. A plateau or final depolarization
4. Final repolarization.

1. *Initial Depolarization*

Initial depolarization is very rapid and it lasts for about 2 msec (0.002 sec). Amplitude of depolarization is about + 20 mV (Fig. 90.1).

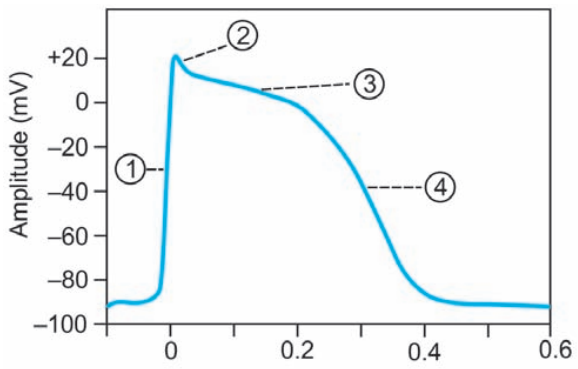


FIGURE 90.1: Action potential in ventricular muscle. 1 = Depolarization, 2 = Initial rapid repolarization, 3 = Plateau, 4 = Final repolarization.

2. *Initial Repolarization*

Immediately after depolarization, there is an initial rapid repolarization for a short period of about 2 msec. The end of rapid repolarization is represented by a notch.

3. *Plateau or Final Depolarization*

Afterwards, the muscle fiber remains in depolarized state for sometime before further repolarization. It forms the plateau (stable period) in action potential curve. The plateau lasts for about 200 msec in atrial muscle fibers and for about 300 msec in ventricular muscle fibers. Due to long plateau in action potential, the contraction time is also longer in cardiac muscle by 5 to 15 times than in skeletal muscle.

4. *Final Repolarization*

Final repolarization occurs after the plateau. It is a slow process and it lasts for about 50 to 80 msec before the re-establishment of resting membrane potential.

■ IONIC BASIS OF ACTION POTENTIAL

1. *Initial Depolarization*

Initial depolarization (first phase) is because of rapid opening of fast **sodium channels** and the **rapid influx of sodium ions,** as in the case of skeletal muscle fiber.

2. *Initial Repolarization*

Initial repolarization is due to the transient (short duration) opening of **potassium channels** and **efflux** of a small quantity of **potassium ions** from the muscle fiber. Simultaneously, the fast sodium channels close suddenly and slow sodium channels open, resulting in **slow influx** of low quantity of **sodium ions.**

3. *Plateau or Final Depolarization*

Plateau is due to the slow opening of **calcium channels.** These channels are kept open for a longer period and cause influx of large number of **calcium ions.** Already the slow sodium channels are opened, through which slow influx of sodium ions continues. Because of the entry of calcium and sodium ions into the muscle fiber, positivity is maintained inside the muscle fiber, producing prolonged depolarization, i.e. plateau. Calcium ions entering the muscle fiber play an important role in the contractile process.

4. *Final Repolarization*

Final repolarization is due to **efflux of potassium ions.** Number of potassium ions moving out of the muscle fiber exceeds the number of calcium ions moving in. It makes negativity inside, resulting in final repolarization. Potassium efflux continues until the end of repolarization.

Restoration of Resting Membrane Potential

At the end of final repolarization, all sodium ions, which had entered the cell throughout the process of action potential move out of the cell and potassium ions move into the cell, by activation of **sodium-potassium pump.** Simultaneously, excess of calcium ions, which had entered the muscle fiber also move out through sodium-calcium pump. Thus, the resting membrane potential is restored.

■ SPREAD OF ACTION POTENTIAL THROUGH CARDIAC MUSCLE

Action potential spreads through cardiac muscle very rapidly because of the presence of gap junctions between the cardiac muscle fibers. Gap junctions are permeable junctions and allow free movement of ions and so the action potential spreads rapidly from one muscle fiber to another fiber.

Action potential is transmitted from atria to ventricles through the fibers of specialized conductive system, which is explained later in this chapter.

■ RHYTHMICITY

■ DEFINITION

Rhythmicity is the ability of a tissue to produce its own impulses regularly. It is also called **autorhythmicity** or

self-excitation. Property of rhythmicity is present in all the tissues of heart. However, heart has a specialized excitatory structure, from which the discharge of impulses is rapid. This specialized structure is called pacemaker. From here, the impulses spread to other parts through the specialized conductive system.

■ PACEMAKER

Pacemaker is the structure of heart from which the impulses for heartbeat are produced. It is formed by the **pacemaker cells** called **P cells.** In mammalian heart, the pacemaker is sinoatrial node (SA node). It was Lewis Sir Thomas, who named SA node as pacemaker of heart, in 1918.

Sinoatrial Node

Sinoatrial (SA) node is a small strip of modified cardiac muscle, situated in the superior part of lateral wall of right atrium, just below the opening of superior vena cava. The fibers of this node do not have contractile elements. These fibers are continuous with fibers of atrial muscle, so that the impulses from the SA node spread rapidly through atria.

Other parts of heart such as atrioventricular (AV) node, atria and ventricle also can produce the impulses and function as pacemakers. Still, SA node is called the pacemaker because the rate of production of impulse (rhythmicity) is more in SA node than in other parts. It is about 70 to 80/minute.

Experimental Evidences

Experimental evidences to prove that SA node is the pacemaker in **mammalian heart:**

1. Stimulation of SA node accelerates the heart rate
2. Destruction of SA node causes immediate stoppage of the heartbeat. After sometime, atrioventricular node becomes the pacemaker and starts generating the impulses. So the heart starts beating, but the rate is slow.
3. Local cooling of SA node decreases the heart rate
4. Local warming of SA node increases the heart rate
5. Electrical activity starts first in SA node.

Spread of Impulses from SA Node

Mammalian heart has got a specialized conductive system, by which the impulses from SA node spreads to other parts of the heart (see below).

Rhythmicity of Different Parts of Human Heart

1. SA node : 70 to 80/minute
2. AV node : 40 to 60/minute
3. Atrial muscle : 40 to 60/minute
4. Purkinje fibers : 35 to 40/minute
5. Ventricular muscle : 20 to 40/minute.

Pacemaker in Amphibian Heart

Sinus venosus is the pacemaker in amphibian heart. It is experimentally proved by:

1. Applying Stannius ligatures
2. When sinus venosus is warmed by warm Ringer solution, heart rate increases
3. When sinus venosus is cooled by cold Ringer solution, heart rate decreases
4. Electrical activity starts first in sinus venosus.

Stannius ligature experiment

Stannius ligature experiment was demonstrated by German biologist **Stannius** in a **pithed frog.** Ligature means tying. **Pithing** is a process by which the brain and spinal cord are severed by using a needle, to abolish all the reflex activities during the experiment. Pithed frog is technically dead but some of its organs such as heart, continue to function for some time.

Chest wall of a pithed frog is opened and heart is exposed. A bent pin is fixed at the tip of ventricle and attached to a recording device by means of a thread. After recording the normal heartbeats (**normal cardiogram** or **sinus rhythm**), a ligature is applied between the sinus venosus and right auricle. It is called first Stannius ligature.

When this ligature is applied, the heart stops beating immediately. It is because the impulses produced by sinus venosus cannot be conducted to the other chambers of the heart. However, the sinus contractions are continued. After sometime, auricular muscle becomes the pacemaker and starts producing the impulses for heartbeat, but at a slower rate. Auricular contraction occurs first, followed by ventricular contraction. This rhythm of the heart is called **auriculoventricular rhythm** (Fig. 90.2).

When a second ligature is applied between auricles and ventricle, the heart stops beating again, because impulses from auricles cannot reach the ventricle. After few minutes, the ventricle produces its **own impulses** and starts beating but at a much slower rate. The slow independent ventricular rhythm is called **idioventricular rhythm.** Thus, all the three parts of the heart, sinus venosus, auricular musculature and ventricular musculature have the property of rhythmicity. However, sinus venosus is the pacemaker because it produces the impulses at a faster rate.

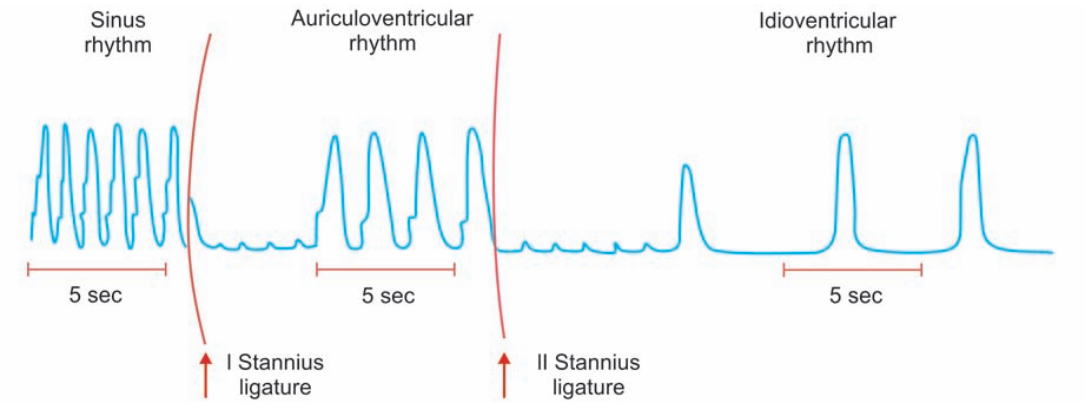


FIGURE 90.2: Effect of Stannius ligatures on frog's heart

Spread of Impulses from Sinus Venosus

Amphibian heart does not have any specialized conductive system. Pacemaker in amphibian heart is the sinus venosus and impulses from **sinus venosus** spreads through the muscles of auricles and ventricle.

Rhythmicity of Different Parts of Amphibian Heart

1. Sinus venosus : 40 to 60/minute
2. Auricular muscle : 20 to 40/minute
3. Ventricular muscle : 15 to 20/minute.

■ ELECTRICAL POTENTIAL IN SA NODE

Resting Membrane Potential – Pacemaker Potential

Pacemaker potential is the unstable resting membrane potential in SA node. It is also called prepotential.

Electrical potential in SA node is different from that of other cardiac muscle fibers. In SA node, each impulse triggers the next impulse. It is mainly due to the unstable resting membrane potential.

Resting membrane potential in SA node has a negativity of –55 to –60 mV. It is different from the negativity of –85 to –95 mV in other cardiac muscle fibers.

Action Potential

Depolarization starts very slowly and the threshold level of –40 mV is reached very slowly. After the threshold level, rapid depolarization occurs up to +5 mV. It is followed by rapid repolarization. Once again, the resting membrane potential becomes unstable and reaches the threshold level slowly (Fig. 90.3).

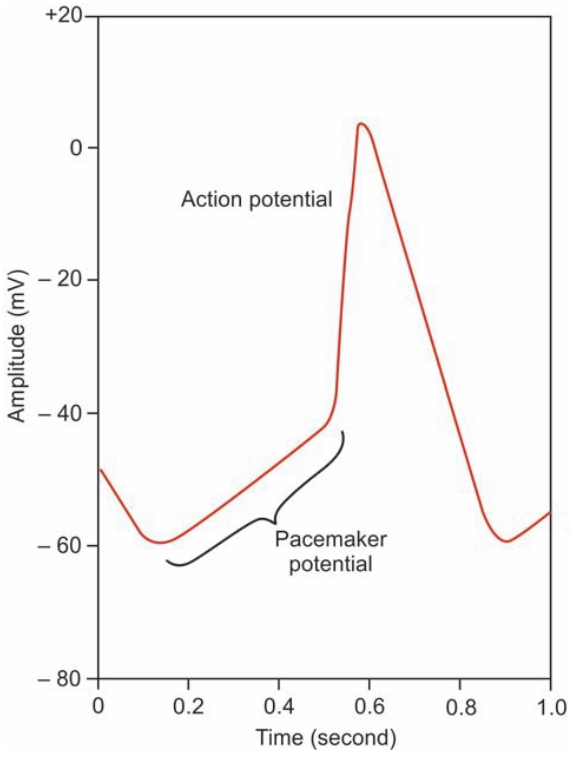


FIGURE 90.3: Pacemaker potential

Ionic Basis of Electrical Activity in Pacemaker

Pacemaker potential or resting membrane potential

Resting membrane potential is not stable in the SA node. To start with, the sodium ions leak into the pacemaker fibers and cause slow depolarization. This

slow depolarization forms the initial part of pacemaker potential. Then, the **calcium channels** start opening. At the beginning, there is a slow **influx of calcium ions** causing further depolarization in the same slower rate. It forms the later part of the pacemaker potential.

Thus, the initial part of pacemaker potential is due to slow **influx of sodium ions** and the later part is due to the slow influx of calcium ions.

Depolarization

When the negativity is decreased to –40 mV, which is the **threshold level,** the action potential starts with rapid depolarization. The depolarization occurs because of influx of more calcium ions. Unlike in other tissues, the depolarization in SA node is mainly due to the influx of calcium ions, rather than sodium ions.

Repolarization

After rapid depolarization, repolarization starts. It is due to the efflux of potassium ions from pacemaker fibers. Potassium channels remain open for a longer time, causing efflux of **more potassium ions.** It leads to the development of more negativity, beyond the level of resting membrane potential. It exists only for a short period. Then, the slow depolarization starts once again, leading to the development of **pacemaker potential,** which triggers the next action potential.

■ CONDUCTIVITY

Human heart has a specialized conductive system, through which impulses from SA node are transmitted to all other parts of the heart (Fig. 90.4).

■ CONDUCTIVE SYSTEM IN HUMAN HEART

Conductive system of the heart is formed by the modified cardiac muscle fibers. These fibers are the specialized cells, which conduct the impulses rapidly from SA node to the ventricles. Conductive tissues of the heart are also called the junctional tissues.

Components of Conductive System in Human Heart

1. AV node
2. Bundle of His
3. Right and left bundle branches
4. Purkinje fibers.

SA node is situated in right atrium, just below the opening of superior vena cava. AV node is situated in right posterior portion of intra-atrial septum. Impulses from SA node are conducted throughout right and left atria. Impulses also reach the AV node via some specialized fibers called internodal fibers.

There are three types of internodal fibers:

1. Anterior internodal fibers of Bachman
2. Middle internodal fibers of Wenckebach
3. Posterior internodal fibers of Thorel.

All these fibers from SA node converge on AV node and interdigitate with fibers of AV node. From AV node, the **bundle of His** arises. It divides into right and left branches, which run on either side of the interventricular septum. From each branch of bundle of His, many **Purkinje fibers** arise and spread all over the ventricular myocardium.

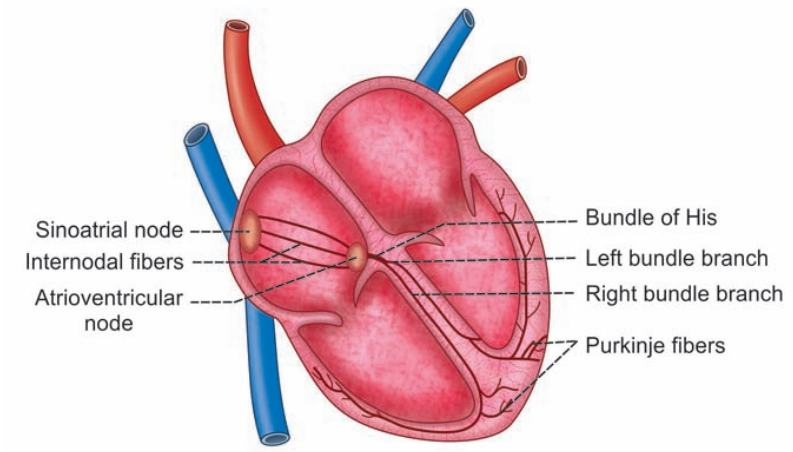


FIGURE 90.4: Sinoatrial node and conductive system of the heart

VELOCITY OF IMPULSES AT DIFFERENT PARTS OF CONDUCTIVE SYSTEM

1. Atrial muscle fibers : 0.3 meter/second
2. Internodal fibers : 1.0 meter/second
3. AV node : 0.05 meter/second
4. Bundle of His : 0.12 meter/second
5. Purkinje fibers : 4.0 meter/second
6. Ventricular muscle fibers : 0.5 meter/second.

Thus, the velocity of impulses is maximum in Purkinje fibers and minimum at AV node.

CONTRACTILITY

Contractility is ability of the tissue to shorten in length (contraction) after receiving a stimulus. Various factors affect the contractile properties of the cardiac muscle. Following are the contractile properties:

ALL-OR-NONE LAW

According to all-or-none law, when a stimulus is applied, whatever may be the strength, the whole cardiac muscle gives maximum response or it does not give any response at all. Below the threshold level, i.e. if the strength of stimulus is not adequate, the muscle does not give response.

All-or-none law is demonstrated in the **quiescent (quiet) heart** of frog. Heart is made quiescent by applying the first Stannius ligature in between the sinus venosus and right auricle.

Ventricle is stimulated by placing the electrode at the base of ventricle.

First, one stimulus is applied with a minimum strength of 1 volt at the base of ventricle and the contraction is recorded. Then, after 20 seconds, the strength of stimulus is increased to 2 volt and the stimulus is applied. The curve is recorded. The procedure is repeated by increasing the strength every time and applying the stimulus with an interval of 20 seconds (Fig. 90.5).

Amplitude of all contractions remains the same, irrespective of increasing the strength of stimulus. This shows that cardiac muscle obeys all-or-none law.

Cause for All-or-none law

All-or-none law is applicable to whole cardiac muscle. It is because of **syncytial** arrangement of cardiac muscle. In the case of skeletal muscle, all-or-none law is applicable only to a single muscle fiber.

STAIRCASE PHENOMENON

When the ventricle of a quiescent heart of frog is stimulated at a short interval of 2 seconds, without changing the strength, the force of contraction increases gradually for the first few contractions and then it remains same. Gradual increase in the force of contraction is called staircase phenomenon.

Cause for Staircase Phenomenon

Staircase phenomenon occurs because of **beneficial effect** (Chapter 30), which facilitates the force of successive contraction. So, there is a gradual increase in force of contraction (Fig. 90.5).

SUMMATION OF SUBLIMINAL STIMULI

When a stimulus with a subliminal strength is applied, the quiescent heart does not show any response. When few stimuli with same subliminal strength are applied in succession, the heart shows response by contraction, due to the summation of stimuli.

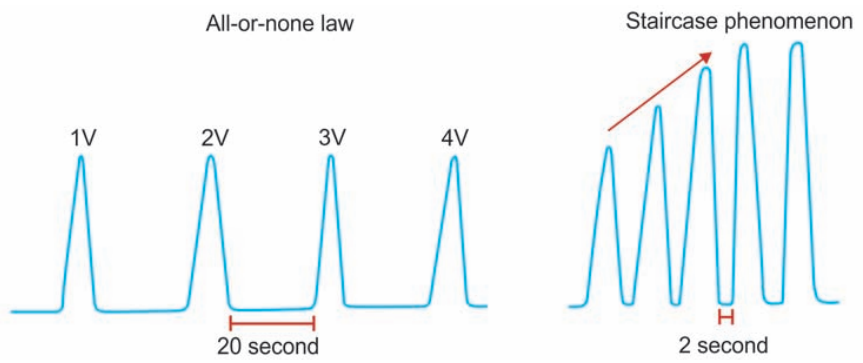


FIGURE 90.5: All-or-none law and staircase phenomenon in cardiac muscle

■ REFRACTORY PERIOD

Refractory period is the period in which the muscle does not show any response to a stimulus. It is of two types:

1. Absolute refractory period
2. Relative refractory period.

Absolute Refractory Period

Absolute refractory period is the period during which the muscle does not show any response at all, whatever may be the strength of the stimulus. It is because, the depolarization occurs during this period. So, a second depolarization is not possible.

Relative Refractory Period

Relative refractory period is the period during which the muscle shows response if the strength of stimulus is increased to maximum. It is the stage at which the muscle is in repolarizing state.

Refractory Period in Skeletal Muscle

In skeletal muscle, the refractory period is short. Absolute refractory period extends during the first half of latent period, measuring about 0.005 sec. Relative refractory period extends during the second half of latent period measuring 0.005 sec. So, the total refractory period is 0.01 sec.

Refractory Period in Cardiac Muscle

Cardiac muscle has a **long refractory period** compared to skeletal muscle. Absolute refractory period extends throughout the contraction period of cardiac muscle and its duration is 0.27 sec. Relative refractory period extends during first half of relaxation period, which is about 0.26 sec. So, the total refractory period is 0.53 sec.

Significance of Long Refractory Period in Cardiac Muscle

Long refractory period in cardiac muscle has three advantages:

1. Summation of contractions does not occur
2. Fatigue does not occur
3. Tetanus does not occur.

Demonstration of Refractory Period in Heart

Refractory period is demonstrated in the heart of a pithed frog. Refractory period can be recorded in beating heart as well as the quiescent heart.

Refractory period in beating heart

First, normal cardiogram is recorded with the heart of a pithed frog. The impulses for heartbeat arise from the sinus venosus. An electrical (external) stimulus is applied by keeping the electrode at the base of the ventricle. When the stimulus is applied during systole, the heart does not show any response. It is because the absolute refractory period extends throughout systole (Fig. 90.6).

When a stimulus is applied during diastole, the heart contracts because, diastole is the relative refractory period. This contraction of the heart is called **extrasystole** or **premature contraction.** Etrasystole is followed by the stoppage of heart in diastole for a while. This diastole is longer than the diastole after regular

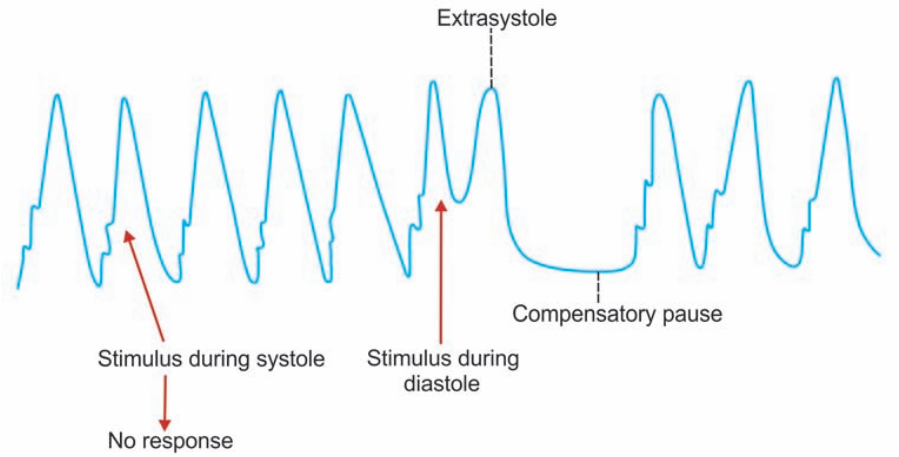


FIGURE 90.6: Refractory period in beating heart of frog

contraction. Temporary stoppage of the heart before it starts contracting is called **compensatory pause.** Duration of extrasystole and compensatory pause is equivalent to the duration of two cardiac cycles.

Cause for compensatory pause

A natural impulse from sinus venosus arrives at the time of contraction period of extrasystole. As this period is absolute refractory period, the natural impulse cannot cause contraction of heart. And the heart has to wait for the arrival of next natural impulse from sinus venosus. Till the arrival of next impulse, the heart stops in diastole.

Refractory period in quiescent heart

Frog's heart is made quiescent by applying the first Stannius ligature. Electrode is placed over the base of ventricle. When two stimuli are applied successively in such a way that the second stimulus falls during contraction period, the heart contracts only once. It is because of the first stimulus. There is no response to second stimulus because systole is the absolute refractory period. However, when a second stimulus is applied during diastole, the heart contracts again and second contraction superimposes over the first one. This shows that the relative refractory period extends during diastole (Fig. 90.7).

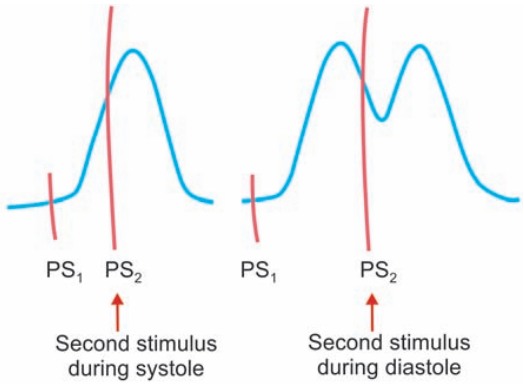


FIGURE 90.7: Refractory period in quiescent heart of a frog. PS_1 = Point of first stimulus, PS_2 = Point of second stimulus.

Cardiac Cycle

Chapter 91

- DEFINITION
- EVENTS
- DIVISIONS AND DURATION
  - ATRIAL EVENTS
  - VENTRICULAR EVENTS
- DESCRIPTION OF ATRIAL EVENTS
  - ATRIAL SYSTOLE
  - ATRIAL DIASTOLE
- DESCRIPTION OF VENTRICULAR EVENTS
  - ISOMETRIC CONTRACTION PERIOD
  - EJECTION PERIOD
  - PROTODIASTOLE
  - ISOMETRIC RELAXATION PERIOD
  - RAPID FILLING PHASE
  - SLOW FILLING PHASE
  - LAST RAPID FILLING PHASE
- INTRA-ATRIAL PRESSURE CHANGES DURING CARDIAC CYCLE
  - SIGNIFICANCE
  - METHODS OF STUDY
  - MAXIMUM AND MINIMUM PRESSURE IN ATRIA
  - INTRA-ATRIAL PRESSURE CURVE
- INTRAVENTRICULAR PRESSURE CHANGES DURING CARDIAC CYCLE
  - SIGNIFICANCE
  - METHODS OF STUDY
  - MAXIMUM AND MINIMUM PRESSURE IN VENTRICLES
  - INTRAVENTRICULAR PRESSURE CURVE
- AORTIC PRESSURE CHANGES DURING CARDIAC CYCLE
  - SIGNIFICANCE
  - METHOD OF STUDY
  - MAXIMUM AND MINIMUM PRESSURE IN AORTA
  - AORTIC PRESSURE CURVE
- VENTRICULAR VOLUME CHANGES DURING CARDIAC CYCLE
  - SIGNIFICANCE
  - METHODS OF STUDY
  - VOLUME OF BLOOD IN RIGHT AND LEFT VENTRICLES
  - VENTRICULAR VOLUME CURVE

DEFINITION

Cardiac cycle is defined as the succession of (sequence of) **coordinated events** taking place in the heart during each beat. Each heartbeat consists of two major periods called systole and diastole. During systole, heart contracts and pumps the blood through arteries. During diastole, heart relaxes and blood is filled in the heart. All these changes are repeated during every heartbeat, in a cyclic manner.

EVENTS OF CARDIAC CYCLE

Events of cardiac cycle are classified into two:

1. Atrial events
2. Ventricular events.

DIVISIONS AND DURATION OF CARDIAC CYCLE

When the heart beats at a normal rate of 72/minute, duration of each cardiac cycle is about 0.8 second.

ATRIAL EVENTS

Atrial events are divided into two divisions:

1. Atrial systole	= 0.11 (0.1) sec
2. Atrial diastole	= 0.69 (0.7) sec.

VENTRICULAR EVENTS

Ventricular events are divided into two divisions:

1. Ventricular systole	= 0.27 (0.3) sec
2. Ventricular diastole	= 0.53 (0.5) sec.

In clinical practice, the term 'systole' refers to ventricular systole and 'diastole' refers to ventricular diastole. Ventricular systole is divided into two subdivisions and ventricular diastole is divided into five subdivisions.

Ventricular Systole

	Time (second)
1. Isometric contraction	= 0.05
2. Ejection period	= 0.22
	0.27

Ventricular Diastole

1. Protodiastole	= 0.04
2. Isometric relaxation	= 0.08
3. Rapid filling	= 0.11
4. Slow filling	= 0.19
5. Last rapid filling	= 0.11
	0.53

Among the atrial events, atrial systole occurs during the last phase of ventricular diastole. Atrial diastole is not considered as a separate phase, since it coincides with the whole of ventricular systole and earlier part of ventricular diastole.

DESCRIPTION OF ATRIAL EVENTS

ATRIAL SYSTOLE

Atrial systole is also known as **last rapid filling phase** or **presystole.** It is usually considered as the last phase of ventricular diastole. Its duration is 0.11 second.

During this period, only a small amount, i.e. 10% of blood is forced from atria into ventricles. Atrial systole is not essential for the maintenance of circulation. Many persons with atrial fibrillation survive for years, without suffering from circulatory insufficiency. However, such persons feel difficult to cope up with physical stress like exercise.

Pressure and Volume Changes

During atrial systole, the intra-atrial pressure increases. Intraventricular pressure and ventricular volume also increase but slightly.

Fourth Heart Sound

Contraction of atrial musculature causes the production of fourth heart sound.

ATRIAL DIASTOLE

After atrial systole, the atrial diastole starts. Simultaneously, ventricular systole also starts. Atrial diastole lasts for about 0.7 sec (accurate duration is 0.69 sec). This long atrial diastole is necessary because, this is the period during which atrial filling takes place. Right atrium receives deoxygenated blood from all over the body through superior and inferior venae cavae. Left atrium receives oxygenated blood from lungs through pulmonary veins.

Atrial Events Vs Ventricular Events

Out of 0.7 sec of atrial diastole, first 0.3 sec (0.27 sec accurately) coincides with ventricular systole. Then, ventricular diastole starts and it lasts for about 0.5 sec (0.53 sec accurately). Later part of atrial diastole coincides with ventricular diastole for about 0.4 sec. So, the heart relaxes as a whole for 0.4 sec. Figure 91.1 shows the correlation between atrial and ventricular events of cardiac cycle.

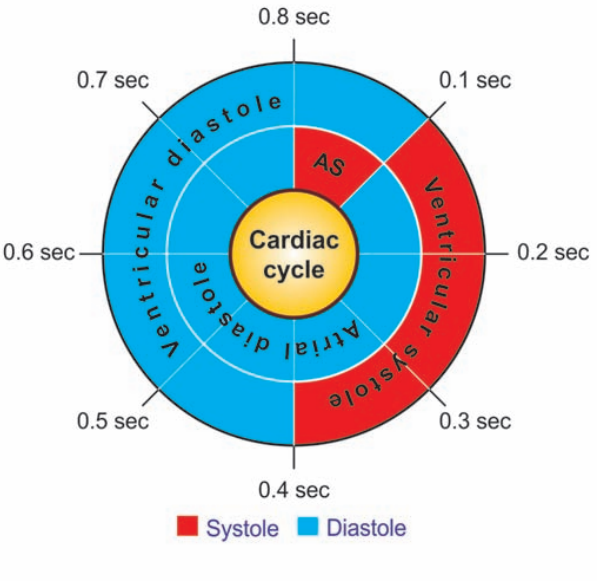


FIGURE 91.1: Atrial and ventricular events of cardiac cycle

DESCRIPTION OF VENTRICULAR EVENTS

ISOMETRIC CONTRACTION PERIOD

Isometric contraction period in cardiac cycle is the first phase of ventricular systole. It lasts for 0.05 second. Isometric contraction is the type of muscular contraction characterized by increase in tension, without any change in the length of muscle fibers. Isometric contraction of ventricular muscle is also called **isovolumetric contraction.**

Immediately after atrial systole, the atrioventricular valves are closed due to increase in ventricular pressure. Semilunar valves are already closed. Now, ventricles contract as closed cavities, in such a way that there is no change in the volume of ventricular chambers or in the length of muscle fibers. Only the tension increases in ventricular musculature.

Because of increased tension in ventricular musculature during isometric contraction, the pressure increases sharply inside the ventricles.

First Heart Sound

Closure of atrioventricular valves at the beginning of this phase produces first heart sound.

Significance of Isometric Contraction

During isometric contraction period, the ventricular pressure increases greatly. When this pressure increases above the pressure in the aorta and pulmonary artery, the semilunar valves open. Thus, the pressure rise in ventricle, caused by isometric contraction is responsible for the **opening of semilunar valves,** leading to ejection of blood from the ventricles into aorta and pulmonary artery.

EJECTION PERIOD

Due to the opening of semilunar valves and isotonic contraction of ventricles, blood is ejected out of both the ventricles. Hence, this period is called ejection period. Duration of this period is 0.22 second. Ejection period is of two stages:

1. *First Stage or Rapid Ejection Period*

First stage starts immediately after the opening of semilunar valves. During this stage, a large amount of blood is rapidly ejected from both the ventricles. It lasts for 0.13 second.

2. *Second Stage or Slow Ejection Period*

During this stage, the blood is ejected slowly with much less force. Duration of this period is 0.09 second.

End-systolic Volume

Ventricles are not emptied at the end of ejection period and some amount of blood remains in each ventricle. Amount of blood remaining in ventricles at the end of ejection period (i.e. at the end of systole) is called end-systolic volume. It is 60 to 80 mL per ventricle.

Measurement of end-diastolic volume

End-systolic volume is measured by **radionuclide angiocardiography** (multigated acquisition – **MUGA scan**) and **echocardiography.** It is also measured by cardiac **catheterization,** computed tomography **(CT) scan** and magnetic resonance imaging **(MRI)** (Chapter 109).

Ejection Fraction

Ejection fraction refers to the fraction (or portion) of end-diastolic volume (see below) that is ejected out by each ventricle per beat. From 130 to 150 mL of end-diastolic volume, 70 mL is ejected out by each ventricle (stroke volume). Normal ejection fraction is 60% to 65%.

Determination of ejection fraction

Ejection fraction (E_f) is the stroke volume divided by end-diastolic volume expressed in percentage. Stroke volume (SV) is, end-diastolic volume (EDV) minus end-systolic volume (ESV).

Ejection fraction is calculated as:

$$E_f = \frac{SV}{EDV} = \frac{EDV - ESV}{EDV}$$

Where,
E_f = Ejection fraction
SV = Stroke volume
EDV = End-diastolic volume
ESV = End-systolic volume.

Significance of determining ejection fraction

Ejection fraction is the measure of left ventricular function. Clinically, it is considered as an important index for assessing the ventricular contractility. Ejection fraction decreases in **myocardial infarction** and **cardiomyopathy.**

■ PROTODIASTOLE

Protodiastole is the first stage of ventricular diastole, hence the name protodiastole. Duration of this period is 0.04 second. Due to the ejection of blood, the pressure in aorta and pulmonary artery increases and pressure in ventricles drops.

When intraventricular pressure becomes less than the pressure in aorta and pulmonary artery, the semilunar valves close. Atrioventricular valves are already closed (see above). No other change occurs in the heart during this period. Thus, protodiastole indicates only the end of systole and beginning of diastole.

Second Heart Sound

Closure of semilunar valves during this phase produces second heart sound.

■ ISOMETRIC RELAXATION PERIOD

Isometric relaxation is the type of muscular relaxation, characterized by decrease in tension without any change in the length of muscle fibers. Isometric relaxation of ventricular muscle is also called **isovolumetric relaxation.**

During isometric relaxation period, once again all the valves of the heart are closed (Fig. 91.2). Now, both the ventricles relax as closed cavities without any change in volume or length of the muscle fiber. Intraventricular pressure decreases during this period. Duration of isometric relaxation period is 0.08 second.

Significance of Isometric Relaxation

During isometric relaxation period, the ventricular pressure decreases greatly. When the ventricular pressure becomes less than the pressure in the atria, the atrioventricular valves open. Thus, the fall in pressure in the ventricles, caused by isometric relaxation is responsible for the **opening of atrioventricular valves,** resulting in filling of ventricles.

■ RAPID FILLING PHASE

When atrionventricular valves are opened, there is a sudden rush of blood (which is accumulated in atria during atrial diastole) from atria into ventricles. So, this period is called the first rapid filling period. Ventricles also relax isotonically. About 70% of filling takes place during this phase, which lasts for 0.11 second.

Third Heart Sound

Rushing of blood into ventricles during this phase causes production of third heart sound.

■ SLOW FILLING PHASE

After the sudden rush of blood, the ventricular filling becomes slow. Now, it is called the slow filling. It is also called **diastasis.** About 20% of filling occurs in this phase. Duration of slow filling phase is 0.19 second.

■ LAST RAPID FILLING PHASE

Last rapid filling phase occurs because of atrial systole. After slow filling period, the atria contract and push a small amount of blood into ventricles. About 10% of ventricular filling takes place during this period. Flow of additional amount of blood into ventricle due to atrial systole is called **atrial kick.**

End-diastolic Volume

End-diastolic volume is the amount of blood remaining in each ventricle at the end of diastole. It is about 130 to 150 mL per ventricle.

Measurement of end-diastolic volume

End-diastolic volume is measured by the same methods, which are used to measure end-systolic volume (see above).

■ INTRA-ATRIAL PRESSURE CHANGES DURING CARDIAC CYCLE

■ SIGNIFICANCE

Pressure in the atria is called the intra-atrial pressure. Intra-atrial pressure is responsible for opening of the atrioventricular valves and ventricular filling. It is also the main factor for the development of venous pulse.

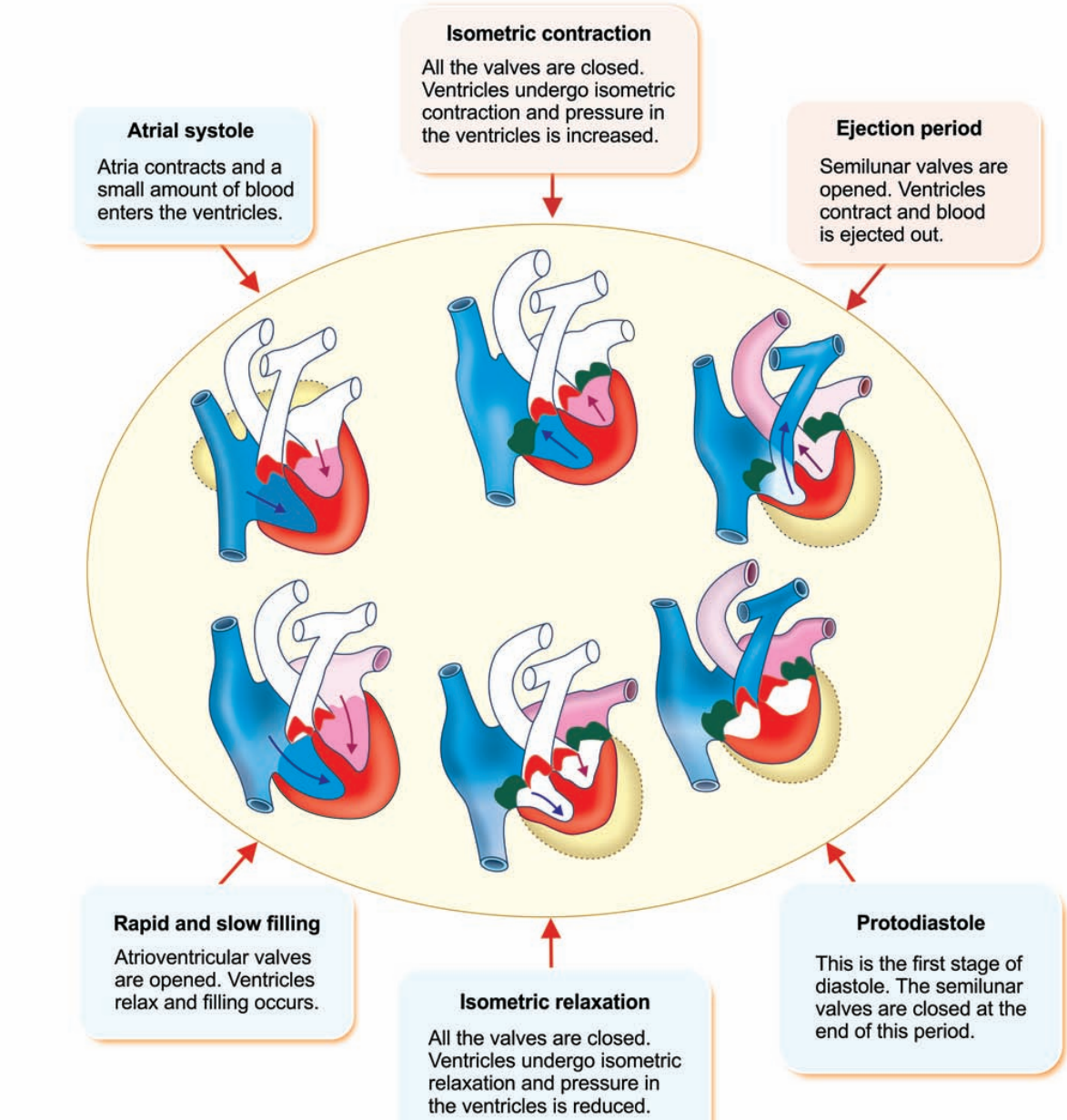

Isometric contraction
All the valves are closed. Ventricles undergo isometric contraction and pressure in the ventricles is increased.
Atrial systole
Atria contracts and a small amount of blood enters the ventricles.
Ejection period
Semilunar valves are opened. Ventricles contract and blood is ejected out.
Rapid and slow filling
Atrioventricular valves are opened. Ventricles relax and filling occurs.
Isometric relaxation
All the valves are closed. Ventricles undergo isometric relaxation and pressure in the ventricles is reduced.
Protodiastole
This is the first stage of diastole. The semilunar valves are closed at the end of this period.

FIGURE 91.2: Events of cardiac cycle

■ METHODS OF STUDY

Right atrial pressure is recorded directly by cardiac **catheterization** (Chapter 98). Left atrial pressure is determined indirectly by measuring pulmonary capillary wedge pressure, which reflects the left atrial pressure accurately.

Pulmonary Capillary Wedge Pressure

Pulmonary capillary wedge pressure is the pressure exerted in the pulmonary capillary bed after obstructing the proximal part of pulmonary artery.

Pulmonary capillary wedge pressure is measured by using a **balloon-tipped multilumen cardiac catheter**

(Swan-Ganz catheter). Tip of the catheter is not open but a pressure transducer is attached to it.

By means of venous puncture, the catheter is guided through right atrium into right ventricle. From the right ventricle, it is advanced towards the proximal portion of pulmonary artery and the balloon is inflated with air by using a syringe. This occludes the pulmonary artery. Then, the catheter alone is advanced further into distal portion of pulmonary artery, leaving the inflated balloon at the proximal portion. It allows the catheter to float in a wedge position. Now the pressure existing in the pulmonary capillary bed ahead of catheter is called pulmonary capillary wedge pressure (the word wedge refers to being obstructed).

When the proximal part of pulmonary artery is obstructed, pressure in the distal part falls rapidly and after about 10 seconds, it becomes equal to left atrial pressure. It is because of the absence of any valve between pulmonary capillary bed and left atrium. So, the left atrial pressure can be determined by measuring pulmonary capillary wedge pressure.

■ MAXIMUM AND MINIMUM PRESSURE IN ATRIA

Maximum and minimum pressures in the left and right atria are given in Table 91.1.

TABLE 91.1: Pressure changes during cardiac cycle

Area	Maximum pressure	Minimum pressure
Left atrium	7 to 8 mm Hg	0 to 2 mm Hg
Right atrium	5 to 6 mm Hg	0 to 2 mm Hg
Left ventricle	120 mm Hg	5 mm Hg
Right ventricle	25 mm Hg	2 to 3 mm Hg
Systemic aorta	120 mm Hg	80 mm Hg
Pulmonary artery	25 mm Hg	7 to 8 mm Hg

■ INTRA-ATRIAL PRESSURE CURVE

Intra-atrial pressure curve is similar to the tracing of jugular venous pulse, which is known as **phlebogram.** It has three positive waves, a, c and v and three negative waves, x, x_1 and y (Fig. 91.3).

'a' Wave

'a' wave is the first positive wave and occurs during **atrial systole.** The pressure rises sharply up to 5 mm Hg in right atrium and 7 mm Hg in left atrium. After reaching the peak, the pressure starts decreasing.

'x' Wave

'x' wave is the first negative wave and appears during the onset of **atrial diastole.** Because of relaxation of atria, the pressure falls. Atrioventricular valves close at the end of this wave.

'c' Wave

'c' wave is the second positive wave and this appears during **isometric contraction.** Rise in pressure is due to

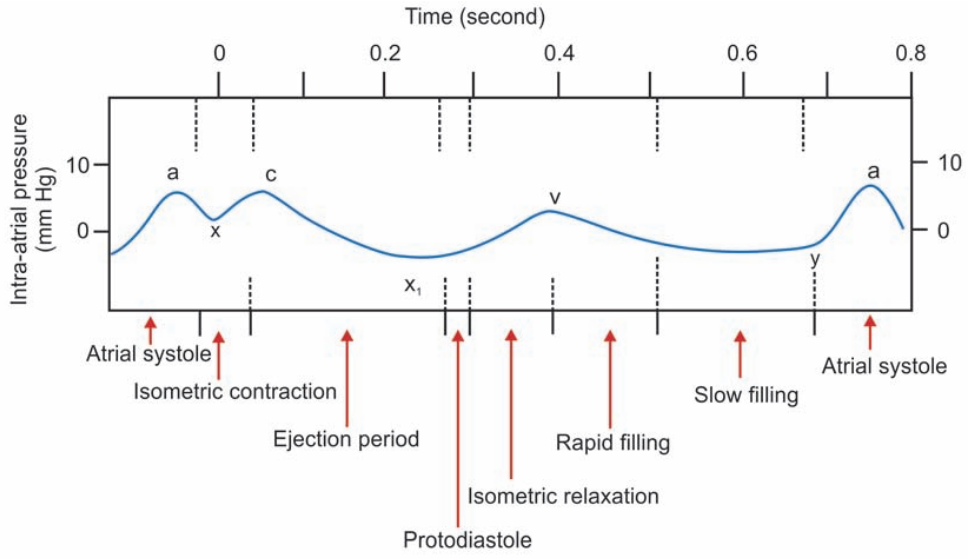


FIGURE 91.3: Intra-arterial pressure changes during cardiac cycle.
a, c, v = Positive waves. x, x_1, y = Negative waves.

the **closure of atrioventricular valves** and the increased intraventricular pressure.

When atrioventricular valves close, there is a little back flow of blood towards atria. When the intraventricular pressure increases, there is bulging of AV valves into the atria. Because of these two factors, the atrial pressure rises.

'x_1' Wave

'x_1' wave is the second negative wave and appears during **ejection period.** During ejection period, the contraction of ventricular musculature pulls the atrioventricular ring towards the ventricles. This causes fall in atrial pressure.

'v' Wave

'v' wave is the third positive wave, which is obtained during **atrial diastole.** It shows a gradual increase in atrial pressure due to filling of blood in atria (venous return).

'y' Wave

'y' wave is the third negative wave and appears after the **opening of AV valves** when the blood rushes from atria into ventricles. So, the pressure in the atria falls.

■ INTRAVENTRICULAR PRESSURE CHANGES DURING CARDIAC CYCLE

■ SIGNIFICANCE

Intraventricular pressure is the pressure developed inside the ventricles of the heart. It is essential for the circulation of blood, because the flow of blood through systemic and pulmonary circulation depends upon the pressure at which the blood is pumped out of ventricles. Thus, intraventricular pressure is essential for the circulation of blood.

■ METHODS OF STUDY

Intraventricular pressure is measured by cardiac catheterization.

■ MAXIMUM AND MINIMUM PRESSURE IN VENTRICLES

There is some difference in the pressure in right ventricle and left ventricle. The pressure is always more in left ventricle than in the right ventricle. Maximum and minimum pressures in the ventricles are given in Table 91.1.

■ INTRAVENTRICULAR PRESSURE CURVE

Intraventricular pressure curve has seven segments (Fig. 91.4).

'A-B' Segment

'A-B' segment is a positive wave and appears during **atrial systole.** Rise in pressure during this period is due to the entry of a small amount of blood into the ventricles because of atrial systole. The pressure rises to about 6 to 7 mm Hg in the right ventricle and to about 7 to 8 mm Hg in the left ventricle.

'B' indicates the **closure of atrioventricular valves.**

'B-C' Segment

'B-C' segment appears during **isometric contraction.** During isometric contraction period, there is a sharp rise in the intraventricular pressure.

'C' denotes the **opening of semilunar valves.**

'C-D' Segment

'C-D' segment appears during **ejection period.** During ejection period, the pressure in the ventricles rises to the peak and then falls down. First part of the curve indicates the maximum ejection and the pressure increases to the maximum. Second part of the curve represents the slow ejection phase when the pressure decreases.

Maximum pressure rise in right ventricle is about 25 mm Hg and the maximum pressure rise in left ventricle is about 120 mm Hg, during the peak of this wave. Maximum pressure in the left ventricle is 4 to 5 times more than that in the right ventricle, because of the thick wall of the left ventricle.

'D-E' Segment

'D-E' segment appears during **protodiastole.** Pressure decreases slightly due to the starting of ventricular relaxation.

'E' indicates the **closure of semilunar valves.**

'E-F' Segment

'E-F' segment is obtained during **isometric relaxation.** There is a sharp fall in the intraventricular pressure during this phase. Pressure in the ventricle falls below the pressure in the atria and this causes the opening of atrioventricular valves.

'F' represents the **opening of atrioventricular valves.**

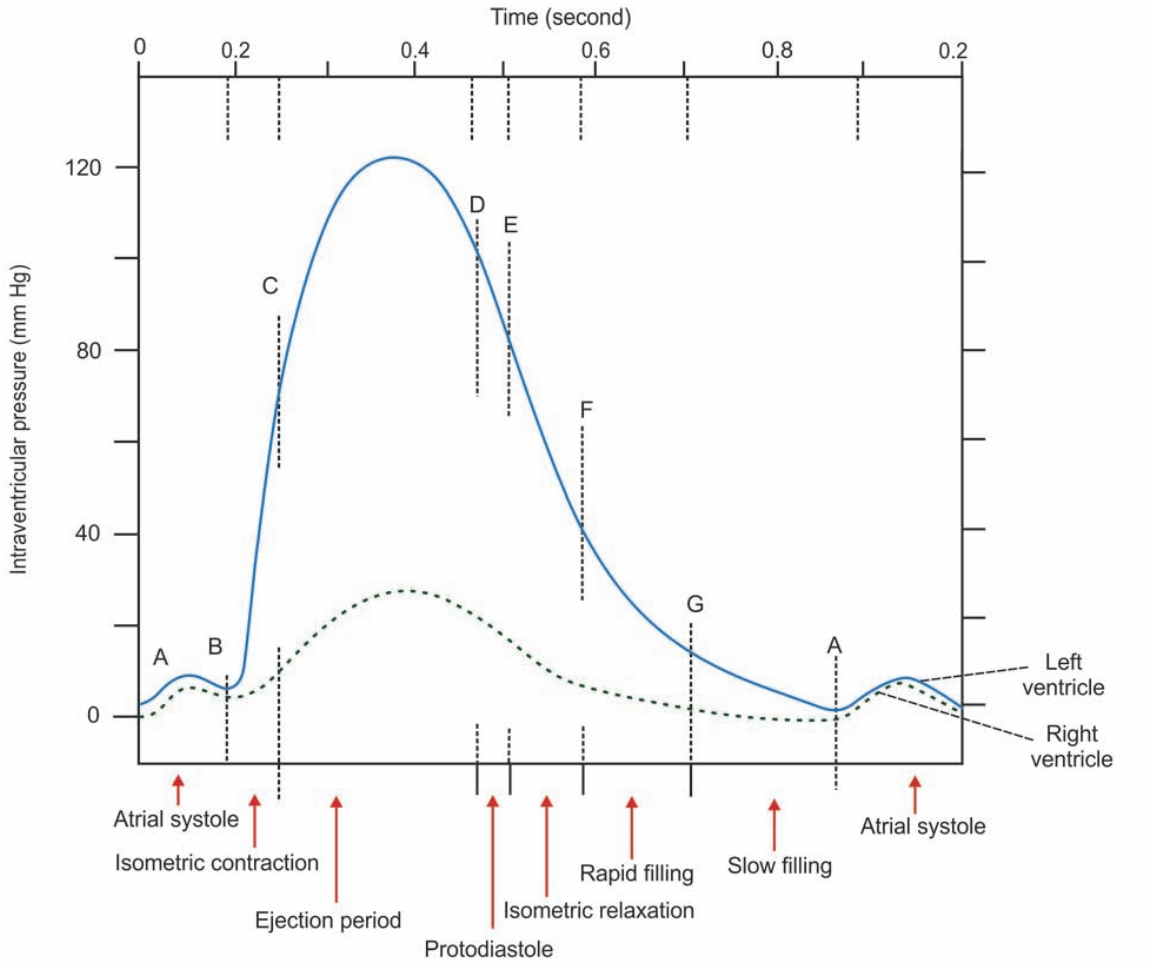


FIGURE 91.4: Intraventricular pressure changes during cardiac cycle

'F-G' Segment

'F-G' segment appears during **rapid filling phase.** In spite of filling of blood, pressure decreases in the ventricles. It is because of the relaxation of the ventricles.

'G-A' Segment

'G-A' segment is the last part of intraventricular pressure curve. It is obtained during **slow filling phase.** Because of continuous relaxation of ventricles during slow filling period, the ventricular pressure decreases further.

AORTIC PRESSURE CHANGES DURING CARDIAC CYCLE

SIGNIFICANCE

Aortic pressure is the pressure developed in the aorta. It is necessary to maintain the blood flow through the circulatory system.

METHOD OF STUDY

Changes in aortic pressure during the cardiac cycle are recorded by using **catheter.**

MAXIMUM AND MINIMUM PRESSURE IN AORTA

Pressure in systemic aorta is always higher than that of pulmonary artery. It is because of the higher pressure in left ventricle than in the right ventricle. Maximum and minimum pressures in aorta are given in Table 91.1. Minimum pressure in systemic aorta is much greater than the minimum pressure in the left ventricle. It is due to the presence of elastic tissues in the aorta, which enable the aorta to recoil and maintain the minimum pressure at a higher level.

AORTIC PRESSURE CURVE

During the ejection period of the cardiac cycle, the pressure in the aorta increases and reaches the peak.

During diastole, it reduces gradually and reaches the minimum level. At the time of closure of semilunar valves, an incisura occurs due to back flow of some blood towards the ventricles (Fig. 91.5).

■ VENTRICULAR VOLUME CHANGES DURING CARDIAC CYCLE

■ SIGNIFICANCE

Volume of blood in the ventricles is an important factor to maintain cardiac output and blood circulation.

■ METHODS OF STUDY

1. *By using Henderson Cardiometer*

This study is done only in animals. Cardiometer is a cup-shaped device with an outlet. At the top, it is closed by means of a rubber diaphragm. A small hole is made in the diaphragm, through which the ventricles of the animal are pushed. Cardiometer is connected to a recording device like **Marey tambour** (a small stainless steel capsule covered by rubber membrane) or **polygraph,** to record the volume changes (Fig. 99.1).

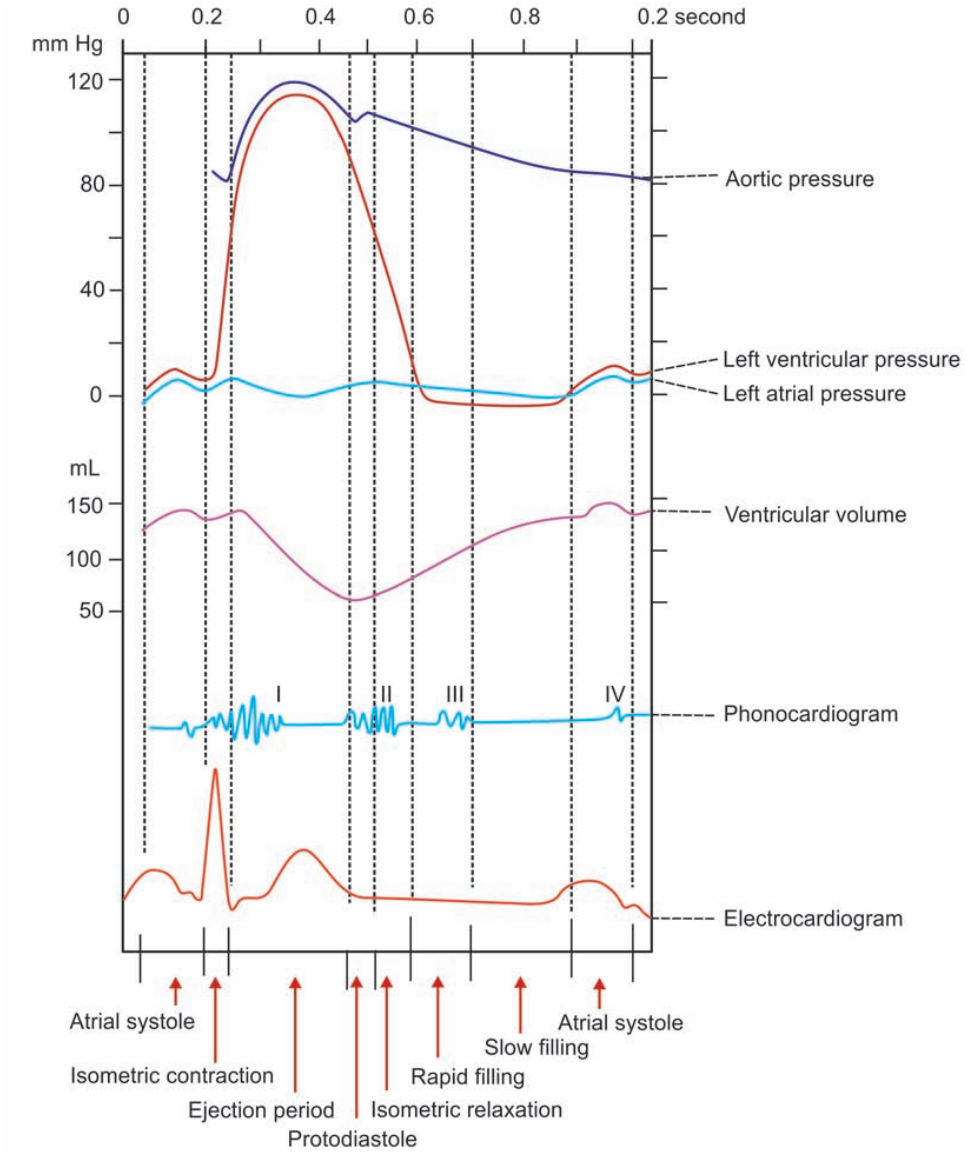


FIGURE 91.5: Comprehensive diagram showing ECG, phonocardiogram, pressure changes and volume changes during cardiac cycle

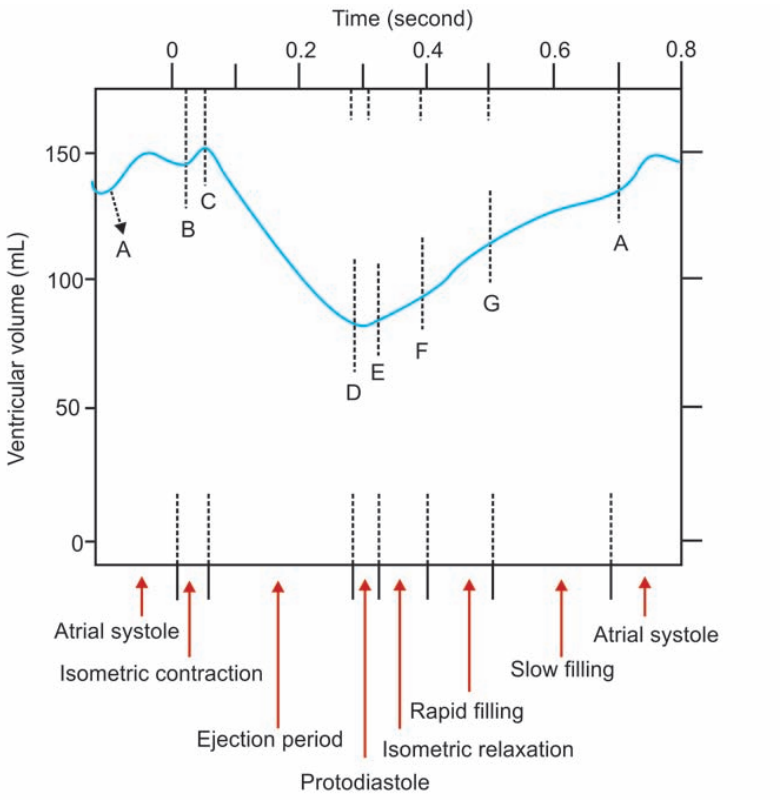


FIGURE 91.6: Ventricular volume changes during cardiac cycle

2. *By Angiography*

Angiography is the **radiographic study** of heart and blood vessels using a **radiopaque contrast** medium. During angiography, it is possible to measure the ventricular dimensional area and thickness of ventricular wall. From the values obtained, the ventricular volume is calculated.

■ VOLUME OF BLOOD IN RIGHT AND LEFT VENTRICLES

End-diastolic Volume and End-systolic Volume

Amount of blood is the same in both right and left ventricles. Maximum volume of blood in each ventricle after filling (end-diastolic volume) is 130 to 150 mL. Minimum volume of blood left in the ventricles at the end of ejection period (end of systolic volume) is 60 to 80 mL.

See above for measurement of end-diastolic volume and end-systolic volume.

Ejection Fraction

Ejection fraction (E_f) is the stroke volume divided by end-diastolic volume, expressed in percentage. See above for determination and significance of determining ejection period.

■ VENTRICULAR VOLUME CURVE

Ventricular volume curve recorded by using **Henderson cardiometer** has seven segments (Fig. 91.6).

'A-B' Segment

'A-B' segment wave is because of **atrial systole** or last filling phase of ventricles, during which a small amount of blood enters the ventricles from the atria. It increases the ventricular volume slightly.

'B' indicates the **closure of atrioventricular valves.**

'B-C' Segment

'B-C' segment is a positive wave, which is obtained during **isometric contraction.** Actually, the ventricular

volume is not altered during isometric contraction. However, the slight upward deflection of this wave is an artifact. It is because the heart thrusts itself into the cardiometer during isometric contraction.

'C' represents the **opening of semilunar valves.**

'C-D' Segment

'C-D' segment occurs during **ejection period.** Initially, there is a sharp fall in the ventricular volume. This occurs during rapid ejection. Later, during slow ejection period, the blood leaves the ventricles slowly. So the ventricular volume decreases slowly.

'D-E' Segment

'D-E' segment part of the ventricular volume curve is recorded during **protodiastole.** There is no change in the ventricular volume during protodiastole.

'E' denotes the closure of semilunar valves.

'E-F' Segment

'E-F' segment appears during **isometric relaxation period** of the cardiac cycle. Actually, the ventricular volume is not altered during isometric relaxation. However, there is a slight upward deflection in the curve due to artifact. It is because of the entrance of blood into coronary artery from aorta during this period. It increases the pressure within the cardiometer.

'F' indicates the **opening of atrioventricular valves.**

'F-G' Segment

'F-G' segment appears during **rapid filling phase.** Rapid rise in ventricular volume is due to sudden rush of blood after the opening of atrioventricular valves.

'G-A' Segment

'G-A' segment is recorded during **slow filling phase.** Ventricular volume increases slowly because of slow filling.

Heart Sounds

Chapter 92

- INTRODUCTION
  - DIFFERENT HEART SOUNDS
  - IMPORTANCE OF HEART SOUNDS
- DESCRIPTION OF DIFFERENT HEART SOUNDS
  - FIRST HEART SOUND
  - SECOND HEART SOUND
  - THIRD HEART SOUND
  - FOURTH HEART SOUND
- TRIPLE AND QUADRUPLE HEART SOUNDS
  - TRIPLE HEART SOUND OR GALLOP RHYTHM
  - QUADRUPLE HEART SOUND
- METHODS OF STUDY OF HEART SOUNDS
  - BY STETHOSCOPE
  - BY MICROPHONE
  - BY PHONOCARDIOGRAM

INTRODUCTION

Heart sounds are the sounds produced by mechanical activities of heart during each cardiac cycle.

Heart sounds are produced by:

1. Flow of blood through cardiac chambers
2. Contraction of cardiac muscle
3. Closure of valves of the heart.

Heart sounds are heard by placing the ear over the chest or by using a stethoscope or microphone. These sounds are also recorded graphically.

DIFFERENT HEART SOUNDS

Four heart sounds are produced during each cardiac cycle:

1. First heart sound
2. Second heart sound
3. Third heart sound
4. Fourth heart sound.

First and second heart sounds are called **classical heart sounds** and are heard by using the stethoscope. These two sounds are more prominent and resemble the spoken words 'LUB, (or LUBB) and 'DUBB' (or DUP), respectively.

Third heart sound is a mild sound and it is not heard by using stethoscope in normal conditions. But it can be heard by using a microphone. Fourth heart sound is an inaudible sound. It becomes audible in pathological conditions only. This sound is studied only by graphic registration, i.e. the phonocardiogram.

IMPORTANCE OF HEART SOUNDS

Study of heart sounds has important diagnostic value in clinical practice because alteration in the heart sounds indicates cardiac diseases involving valves of the heart.

DESCRIPTION OF HEART SOUNDS

FIRST HEART SOUND

First heart sound is produced during **isometric contraction** period and earlier part of **ejection period** (Table 92.1).

TABLE 92.1: Heart sounds

Features	First heart sound	Second heart sound	Third heart sound	Fourth heart sound
Occurs during	Isometric contraction period and part of ejection period	Protodiastole and part of isometric relaxation	Rapid filling phase	Atrial systole
Characteristics	Long, soft and low pitched Resembles the word 'LUBB'	Short, sharp and high pitched Resembles the word 'DUP'	Low pitched	Inaudible sound
Cause	Closure of atrioventricular valves	Closure of semilunar valves	Rushing of blood into ventricle	Contraction of atrial musculature
Duration (sec)	0.10 to 0.17	0.10 to 0.14	0.07 to 0.10	0.02 to 0.04
Frequency (cycles per sec)	25 to 45	50	1 to 6	1 to 4
Relation with ECG	Coincides with peak of 'R' wave	Precedes or appears 0.09 second after peak of 'T' wave	Between 'T' wave and 'P' wave	Between 'P' wave and 'Q' wave
Number of vibrations in phonocardiogram	9 to 13	4 to 6	1 to 4	1 to 2

Causes

Major cause for first heart sound is the sudden and synchronous (simultaneous) closure of **atrioventricular valves.** However, some other factors are also involved. Four types of factors are responsible for the production of the first heart sound.

1. *Valvular factor*

Synchronous closure of atrioventricular valves set up the vibrations in the **valvular leaflets** and **chordae tendineae.** These vibrations are mainly responsible for the production of the first heart sound.

2. *Vascular factor*

Rush of blood from the ventricles into aorta and pulmonary artery during ejection period is also responsible for the production of the first heart sound.

3. *Muscular factor*

Myocardial tension and the contraction of ventricular muscle during isometric contraction and the ejection periods also add to the production of the first heart sound.

4. *Atrial factor*

Vibrations produced by the atrial systole also play a role in the production of the first heart sound.

Characteristics

First heart sound is a long, soft and low-pitched sound. It resembles the spoken word **'LUBB'.** The duration of this sound is 0.10 to 0.17 second. Its frequency is 25 to 45 cycles/second.

Applied Physiology

1. *Reduplication of first heart sound*

Reduplication means splitting of the heart sound. First heart sound is split when the atrioventricular valves do not close simultaneously **(asynchronous closure).** Splitting of first heart sound in normal conditions **(physiological splitting)** is rare. **Pathological splitting** of first heart sound occurs in stenosis of atrioventricular valves and atrial septal defect.

2. *Soft first heart sound*

Heart sound becomes soft when the intensity of sound decreases. A soft first heart sound is heard in low blood pressure, severe heart failure, myocardial infarction and myxedema.

3. *Loud or accentuated first heart sound*

First heart sound becomes louder or accentuated (becoming prominent) in conditions like mitral stenosis, **Wolff-Parkinson-White syndrome** and acute rheumatic fever. It is loud in patients with thin chest wall also.

4. *Cannon sound*

Cannon sound refers to the loud first heart sound that is heard intermittently. It is heard in ventricular tachycardia and complete atrioventricular block.

First Heart Sound and ECG

First heart sound coincides with peak of 'R' wave in ECG.

■ SECOND HEART SOUND

Second heart sound is produced at the end of **protodiastolic period.**

Cause

Second heart sound is produced due to the sudden and synchronous closure of the **semilunar valves.**

Characteristics

Second heart sound is a short, sharp and high-pitched sound. It resembles the spoken word **'DUBB'** (or DUP). Duration of second heart sound is 0.10 to 0.14 second. Its frequency is 50 cycles/second.

Applied Physiology

1. Reduplication of second heart sound

Splitting of second heart sound occurs due to asynchronous closure of semilunar valves. It may occur both in physiological and pathological conditions.

Physiological splitting: It occurs during deep inspiration. Normally, aortic valve closes prior to the closure of pulmonary valve. Interval between the two valves widens during inspiration and narrows during expiration.

Increased negative intrathoracic pressure during deep inspiration increases lung expansion and venous return into right atrium. However, the venous return from lungs to left atrium is reduced during this condition. Because of increased venous return in right atrium and subsequent increase in blood volume in right ventricle, pulmonary valve is kept open for slightly longer time than the aortic valve. So, the pulmonary valve closes little later than the aortic valve causing splitting of second heart sound.

Pathological splitting: The splitting of second heart sound occurs during pulmonary stenosis, right bundle branch block and right ventricular hypertrophy.

Reverse splitting: It is the splitting of second heart sound, in which aortic valve closes after the closure of pulmonary valve. It is due to the delay in emptying of left ventricle. It is also called **paradoxical splitting** (paradoxical = contradictory or opposite). Reverse splitting is common in left bundle-branch block, aortic stenosis and left ventricular hypertrophy.

2. *Loud or accentuated second heart sound*

Loud or accentuated second heart sound is produced by the closure of either aortic valve or pulmonary valve. Aortic valve produces loud sound during systemic hypertension and **coarctation** (narrowing) of aorta. Pulmonary valve produces loud sound during pulmonary hypertension.

3. *Soft second heart sound*

Second heart sound becomes soft in heart failure.

Second Heart Sound and ECG

Second heart sound coincides with the 'T' wave in ECG. Sometimes, it may precede the 'T' wave or it may commence after the peak of 'T' wave.

■ THIRD HEART SOUND

Third heart sound is a low-pitched sound that is produced during **rapid filling period** of the cardiac cycle. It is also called ventricular gallop or protodiastolic gallop, as it is produced during earlier part of diastole.

Usually, the third heart sound is **inaudible** by stethoscope and it can be heard only by using microphone.

Causes

Third heart sound is produced by the **rushing of blood** into ventricles and vibrations set up in the ventricular wall during rapid filling phase. It may also be due to vibrations set up in chordae tendineae.

Characteristics

Third heart sound is a short and low-pitched sound. Duration of this sound is 0.07 to 0.10 second. Its frequency is 1 to 6 cycles/second.

Conditions when Third Heart Sound becomes Audible by Stethoscope

Third heart sound can be heard by stethoscope in children and athletes. Pathological conditions when third heart sound becomes loud and audible by stethoscope are aortic regurgitation, cardiac failure and cardiomyopathy with dilated ventricles.

When third heart sound is heard by stethoscope, the condition is called **triple heart sound** (see below). Third heart sound is usually heard best with the bell of stethoscope placed at the apex beat area, when the patient is in left **lateral decubitus** (lying on left side) position.

Third Heart Sound and ECG

Third heart sound appears between 'T' and 'P' waves of ECG.

■ FOURTH HEART SOUND

Normally, the fourth heart sound is an **inaudible** sound. It becomes audible only in pathological conditions. It is studied only by graphical recording, i.e. by phonocardiography. This sound is produced during **atrial systole** (late diastole) and it is considered as the physiologic atrial sound. It is also called **atrial gallop** or **presystolic gallop.**

Causes

Fourth heart sound is produced by contraction of **atrial musculature** and vibrations are set up in atrial musculature, flaps of the atrioventricular valves during systole. It is also due to the vibrations set up in the ventricular myocardium because of ventricular distention during atrial systole.

Characteristics

Fourth heart sound is a short and low-pitched sound. Duration of this sound is 0.02 to 0.04 second. Its frequency is 1 to 4 cycles/second.

Conditions when Fourth Heart Sound becomes Audible

Fourth heart sound becomes audible by stethoscope when the ventricles become stiff. Ventricular stiffness occurs in conditions like ventricular hypertrophy, long standing hypertension and aortic stenosis. To overcome the ventricular stiffness, the atria contract forcefully, producing audible fourth heart sound.

When fourth heart sound is heard by stethoscope, the condition is called **triple heart sound** (see below). It is usually heard best with the bell of stethoscope placed at the apex beat area, when the patient is in supine or left semilateral position.

Fourth Heart Sound and ECG

Fourth heart sound coincides with the interval between the end of 'P' wave and the onset of 'Q' wave.

■ TRIPLE AND QUADRUPLE HEART SOUNDS

■ TRIPLE HEART SOUND OR GALLOP RHYTHM

Triple heart sound or **triple rhythm** is an **abnormal rhythm** of heart, characterized by three clear heart sounds during each heart beat. It is due to an abnormal third or fourth heart sound that is heard besides first and second heart sounds. It is also called **gallop rhythm,** since it resembles the sound of a horse's gallop. Usually, it is indicative of serious cardiovascular disease.

Conditions when Triple Heart Sound is Produced

Triple heart sound is produced in conditions like myocardial infarction and severe hypertension.

■ QUADRUPLE HEART SOUND

Quadruple heart sound is an abnormal rhythm of heart, characterized by four clear heart sounds during each heart beat. It is also called **quadruple rhythm.** It is due to third and fourth heart sounds that are heard besides first and second heart sounds. It is also called **quadruple gallop.**

Quadruple heart sound is also indicative of serious cardiovascular disease.

Conditions when Quadruple Heart Sound is Produced

Quadruple heart sound is produced in patients with congestive heart failure.

Summation Gallop

Whenever there is tachycardia in patients with quadruple heart sound, the third and fourth heart sounds merge together and give rise to a single sound. This sound is called **summation gallop** and it resembles gallop rhythm.

■ METHODS OF STUDY OF HEART SOUNDS

Heart sounds are studied by three methods:

1. By using stethoscope
2. By using microphone
3. By using phonocardiogram.

■ BY STETHOSCOPE

First and second heart sounds are heard on the auscultation areas, by using the stethoscope. The chest piece of the stethoscope is placed over four areas on the chest, which are called auscultation areas.

Auscultation Areas

i. Mitral area (Bicuspid area)

Mitral area is in the left 5th intercostal space, about 10 cm away from the midline (midclavicular line). Sound produced by the closure of mitral valve (first heart

sound) is transmitted well into this area. It is also called **apex beat area** because apex beat is felt in this area.

Apex beat

Apex beat is the thrust of the apex of ventricles, against the chest wall during systole.

ii. *Tricuspid area*

Tricuspid area is on the xiphoid process. Sound produced by the closure of tricuspid valve (first heart sound) is transmitted well into this area.

iii. *Pulmonary area*

Pulmonary area is on the left 2nd intercostal space, close to sternum. Sound produced by the closure of pulmonary valve (second heart sound) is heard well on this area.

iv. *Aortic area*

Aortic area is over the right 2nd intercostal space, close to the sternum. On this area, the sound produced by the closure of aortic valve (second heart sound) is heard well.

First heart sound is best heard in mitral and tricuspid areas. However, it is heard in other areas also but the intensity is less. Similarly, the second heart sound is best heard in pulmonary and aortic areas. It is also heard in other areas with less intensity.

■ BY MICROPHONE

A highly sensitive microphone is placed over the chest. The heart sounds are amplified by means of an **amplifier** and heard by using a **loudspeaker.** First, second and third heart sounds are heard by this method.

■ BY PHONOCARDIOGRAM

Phonocardiography is the technique used to record the heart sounds. Phonocardiogram is the graphical record of heart sounds. It is done by placing an electronic **sound transducer** over the chest. This transducer is connected to a recording device like polygraph. All the four heart sounds can be recorded in phonocardiogram. It helps to analyze the frequency of the sound waves.

Appearance of Heart Sounds in Phonocardiogram

In phonocardiogram, the heart sounds are recorded in the following manner (Fig. 91.6).

First heart sound

First heart sound is recorded as single group of waves. The waves are of small amplitude to start with. Later, the amplitude rapidly rises and falls to form **crescendo** and **diminuendo** series of waves. About 9 to 13 waves appear.

Second heart sound

Second heart sound appears as single group of waves, which have same amplitude. About 4 to 6 waves are recorded.

Third heart sound

Third heart sound is found in phonocardiogram with only 1 to 4 waves grouped together.

Fourth heart sound

Mostly, the fourth heart sound merges with first heart sound. If it appears as separate form, it has 1 to 2 waves with very low amplitude.

Cardiac Murmur

Chapter 93

- INTRODUCTION
  - CAUSES OF MURMUR
- CLASSIFICATION OF MURMUR
  - SYSTOLIC MURMUR
  - DIASTOLIC MURMUR
  - CONTINUOUS MURMUR

INTRODUCTION

Cardiac murmur is the abnormal or unusual heart sound. It is also called **abnormal heart sound** or **cardiac bruit.** Cardiac murmur is heard by stethoscope, along with normal heart sounds.

Cardiac murmur is heard by placing chest piece of stethoscope over the auscultatory areas. Murmur due to disease of a particular valve is heard well over the auscultatory area of that valve. Sometimes, the murmur is felt by palpation as **'thrills'.** In some patients, murmur is heard without any aid, even at a distance of few feet away from the patient.

CAUSES OF MURMUR

Cardiac murmur is produced because of change in the pattern of blood flow. Normally, blood flows in **streamline** through the heart and blood vessels. However, during abnormal conditions like valvular diseases, the blood flow becomes **turbulent.** It produces the cardiac murmur.

Murmur is produced because of valvular diseases, septal defects and vascular defects (Table 93.1).

Valvular Diseases

Valvular diseases are of two types:

1. Stenosis
2. Incompetence.

1. *Stenosis*

Stenosis means narrowing of heart valve. Blood flows rapidly with turbulence through the narrow orifice of the valve, resulting in murmur.

TABLE 93.1: Causes for cardiac murmur

Type of murmur	Causes
Systolic murmur	1. Incompetence of atrioventricular valves 2. Stenosis of semilunar valves 3. Anemia 4. Septal defect 5. Coarctation of aorta
Diastolic murmur	1. Stenosis of atrioventricular valves 2. Incompetence of semilunar valves
Continuous murmur	1. Patent ductus arteriosus

2. *Incompetence*

Incompetence refers to weakening of the heart valve. When the valve becomes weak, it cannot close properly. It causes back flow of blood, resulting in **turbulence.** This disease is also called **regurgitation** or **valvular insufficiency.**

■ CLASSIFICATION OF MURMUR

Cardiac murmur is classified into three types:

A. Systolic murmur
B. Diastolic murmur
C. Continuous murmur.

■ SYSTOLIC MURMUR

Systolic murmur is the murmur which is produced during systole. It is produced in the following conditions:

1. *Incompetence of Atrioventricular Valves*

When the atrioventricular valves become weak, these valves cannot close completely. It causes **regurgitation** of blood from ventricles to the atria during ventricular systole, producing the murmur. It is a **harsh blowing sound** with high frequency.

2. *Stenosis of Semilunar Valves*

During stenosis of aortic valve, the left ventricular pressure raises up to 300 mm Hg during systole. It causes a greater turbulence in the blood flow. The vibrations of this sound can be felt as **'thrills'** by palpation over lower neck region and upper chest. In severe conditions, the sound is heard even a few feet away from the affected person. It is a **harsh** and a **loud sound.**

3. *Murmur due to Anemia*

A systolic murmur is heard in severe anemia because of reduced viscosity and accelerated flow of blood.

4. *Septal Defect*

During interventricular septal defect, blood flows from left ventricle to right ventricle during systole. It produces a systolic murmur. Septal defect is a rare disorder.

5. *Coarctation of Aorta*

Coarctation of aorta is a congenital disorder, characterized by the narrowing of a part of systemic aorta. A loud murmur is produced during systole and it is heard in the earlier part of diastole also.

■ DIASTOLIC MURMUR

Diastolic murmur is the murmur that is produced during diastole. It is produced in the following conditions:

1. *Stenosis of Atrioventricular Valves*

When the atrioventricular valves become narrow, the turbulence of blood flow occurs during diastole, i.e. when blood enters the ventricles from atria. Murmur due to stenosis of mitral valve is heard better at mitral area. Murmur due to stenosis of tricuspid valve is heard best at tricuspid area. It is a **weak sound** with low frequency.

Sometimes, murmur due to mitral stenosis cannot be heard by stethoscope, due to low frequency. But it can be felt as a mild thrill over mitral area of the chest.

2. *Incompetence of Semilunar Valves*

Murmur is produced during the regurgitation of blood from aorta into the ventricle, through incompetent semilunar valve during diastole. It is like a **blowing sound** with low frequency.

■ CONTINUOUS MURMUR

Continuous murmur is the murmur that is heard in conditions such as patent ductus arteriosus.

Patent Ductus Arteriosus

Intact ductus arteriosus is called patent ductus arteriosus (Chapter 114). A continuous murmur is heard in this condition. However, intensity of the sound is more during systole and less during diastole. Because of this, it is also called **machinery murmur.**

It is a **harsh blowing sound** and is heard best in the pulmonary area. The murmur is heard 1 year after birth.

Electrocardiogram (ECG)

Chapter 94

- DEFINITIONS
- USES OF ECG
- ELECTROCARDIOGRAPHIC GRID
  - DURATION
  - AMPLITUDE
  - SPEED OF THE PAPER
- ECG LEADS
  - BIPOLAR LEADS
  - UNIPOLAR LEADS
- WAVES OF NORMAL ECG
  - 'P' WAVE
  - 'QRS' COMPLEX
  - 'T' WAVE
  - 'U' WAVE
- INTERVALS AND SEGMENTS OF ECG
  - 'P-R' INTERVAL
  - 'Q-T' INTERVAL
  - 'S-T' SEGMENT
  - 'R-R' INTERVAL

DEFINITIONS

Electrocardiography

Electrocardiography is the **technique** by which electrical activities of the heart are studied. The spread of excitation through myocardium produces local electrical potential. This low-intensity current flows through the body, which acts as a **volume conductor.** This current can be picked up from surface of the body by using suitable electrodes and recorded in the form of electrocardiogram. This technique was discovered by Dutch physiologist, **Einthoven Willem,** who is considered the father of electrocardiogram (ECG).

Electrocardiograph

Electrocardiograph is the **instrument** (machine) by which electrical activities of the heart are recorded.

Electrocardiogram

Electrocardiogram (ECG or EKG from electrokardiogram in Dutch) is the record or **graphical registration** of electrical activities of the heart, which occur prior to the onset of mechanical activities. It is the **summed electrical activity** of all cardiac muscle fibers recorded from surface of the body.

USES OF ECG

Electrocardiogram is useful in determining and diagnosing the following:

1. Heart rate
2. Heart rhythm
3. Abnormal electrical conduction
4. Poor blood flow to heart muscle (ischemia)
5. Heart attack

6. Coronary artery disease
7. Hypertrophy of heart chambers.

ELECTROCARDIOGRAPHIC GRID

The paper that is used for recording ECG is called ECG paper. ECG machine amplifies the electrical signals produced from the heart and records these signals on a moving ECG paper.

Electrocardiographic grid refers to the markings (lines) on ECG paper. ECG paper has horizontal and vertical lines at regular intervals of 1 mm. Every 5th line (5 mm) is thickened.

DURATION

Time duration of different ECG waves is plotted horizontally on X-axis.

On X-axis

1 mm = 0.04 second
5 mm = 0.20 second

AMPLITUDE

Amplitude of ECG waves is plotted vertically on Y-axis.

On Y-axis

1 mm = 0.1 mV
5 mm = 0.5 mV

SPEED OF THE PAPER

Movement of paper through the machine can be adjusted by two speeds, 25 mm/second and 50 mm/second. Usually, speed of the paper during recording is fixed at 25 mm/second. If heart rate is very high, speed of the paper is changed to 50 mm/second.

ECG LEADS

ECG is recorded by placing series of electrodes on the surface of the body. These electrodes are called ECG leads and are connected to the ECG machine.

Electrodes are fixed on the limbs. Usually, right arm, left arm and left leg are chosen. Heart is said to be in the center of an **imaginary equilateral triangle** drawn by connecting the roots of these three limbs. This triangle is called Einthoven triangle.

Einthoven Triangle and Einthoven Law

Einthoven triangle is defined as an equilateral triangle that is used as a model of standard limb leads used to record electrocardiogram. Heart is presumed to lie in the center of Einthoven triangle.

Electrical potential generated from the heart appears simultaneously on the roots of the three limbs, namely the left arm, right arm and the left leg.

Refer next Chapter for Einthoven law.

ECG is recorded in 12 leads, which are generally classified into two categories.

I. Bipolar leads
II. Unipolar leads.

BIPOLAR LIMB LEADS

Bipolar limb leads are otherwise known as **standard limb leads.** Two limbs are connected to obtain these leads and both the electrodes are **active recording electrodes,** i.e. one electrode is positive and the other one is negative (Fig. 94.1).

Standard limb leads are of three types:

a. Limb lead I
b. Limb lead II
c. Limb lead III.

Lead I

Lead I is obtained by connecting right arm and left arm. Right arm is connected to the negative terminal of the instrument and the left arm is connected to the positive terminal.

Lead II

Lead II is obtained by connecting right arm and left leg. Right arm is connected to the negative terminal of the instrument and the left leg is connected to the positive terminal.

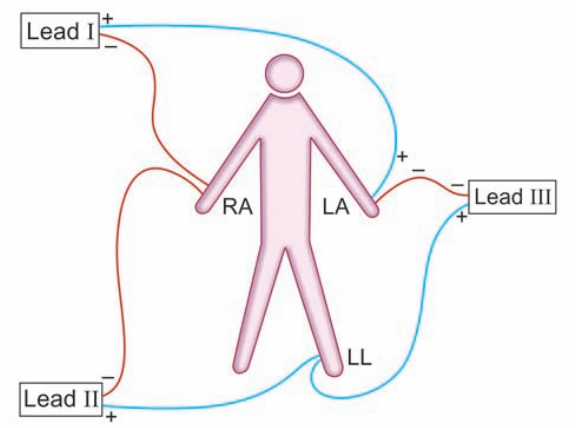


FIGURE 94.1: Position of electrodes for standard limb leads
RA = Right arm, LA = Left arm, LL=Left leg.

Lead III

Lead III is obtained by connecting left arm and left leg. Left arm is connected to the negative terminal of the instrument and the left leg is connected to the positive terminal.

■ UNIPOLAR LEADS

Here, one electrode is **active electrode** and the other one is an **indifferent electrode.** Active electrode is positive and the indifferent electrode is serving as a **composite negative electrode.**

Unipolar leads are of two types:

1. Unipolar limb leads
2. Unipolar chest leads.

1. *Unipolar Limb Leads*

Unipolar limb leads are also called **augmented limb leads** or **augmented voltage leads.** Active electrode is connected to one of the limbs. Indifferent electrode is obtained by connecting the other two limbs through a resistance.

Unipolar limb leads are of three types:

i. aVR lead
ii. aVL lead
iii. aVF lead.

i. *aVR lead*

Active electrode is from right arm. Indifferent electrode is obtained by connecting left arm and left leg.

ii. *aVL lead*

Active electrode is from left arm. Indifferent electrode is obtained by connecting right arm and left leg.

iii. *aVF lead*

Active electrode is from left leg (foot). Indifferent electrode is obtained by connecting the two upper limbs.

2. *Unipolar Chest Leads*

Chest leads are also called **'V' leads** or **precardial chest leads.** Indifferent electrode is obtained by connecting the three limbs, viz. left arm, left leg and right arm, through a **resistance** of 5000 ohms. Active electrode is placed on six points over the chest (Fig. 94.2). This electrode is known as the chest electrode and the six points over the chest are called V_1, V_2, V_3, V_4, V_5 and V_6. V indicates vector, which shows the direction of current flow.

Position of chest leads:

V_1 : Over 4th intercostal space near right sternal margin
V_2 : Over 4th intercostal space near left sternal margin

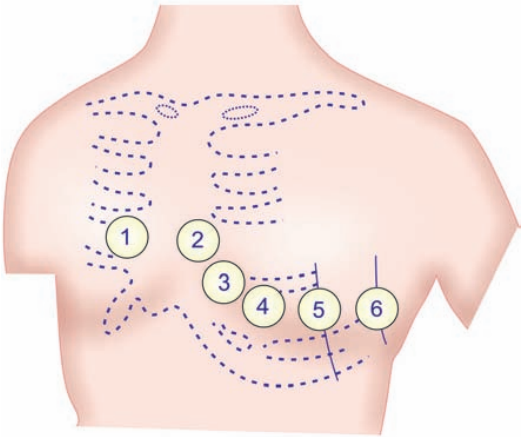


FIGURE 94.2: Position of electrodes for chest leads (V_1 to V_6)

V_3 : In between V_2 and V_4
V_4 : Over left 5th intercostal space on the mid clavicular line
V_5 : Over left 5th intercostal space on the anterior axillary line
V_6 : Over left 5th intercostal space on the mid axillary line.

■ WAVES OF NORMAL ECG

Normal ECG consists of waves, complexes, intervals and segments. Waves of ECG recorded by limb lead II are considered as the typical waves. Normal electrocardiogram has the following waves, namely P, Q, R, S and T (Table 94.1 and Fig. 94.3). Einthoven had named the waves of ECG starting from the middle of the English alphabets (P) instead of starting from the beginning (A).

Major Complexes in ECG

1. 'P' wave, the atrial complex
2. 'QRS' complex, the initial ventricular complex
3. 'T' wave, the final ventricular complex
4. 'QRST', the ventricular complex.

■ 'P' WAVE

'P' wave is a positive wave and the first wave in ECG. It is also called **atrial complex.**

Cause

'P' wave is produced due to the **depolarization** of **atrial musculature.** Depolarization spreads from SA node to all parts of atrial musculature. **Atrial repolarization** is not

TABLE 94.1: Waves of normal ECG

Wave/Segment	From – To	Cause	Duration (second)	Amplitude (mV)
P wave	–	Atrial depolarization	0.1	0.1 to 0.12
QRS complex	Onset of Q wave to the end of S wave	Ventricular depolarization and atrial repolarization	0.08 to 0.10	Q = 0.1 to 0.2 R = I S = 0.4
T wave	–	Ventricular repolarization	0.2	0.3
P-R interval	Onset of P wave to onset of Q wave	Atrial depolarization and conduction through AV node	0.18 (0.12 to 0.2)	–
Q-T interval	Onset of Q wave and end of T wave	Ventricular depolarization and ventricular repolarization	0.4 to 0.42	–
S-T segment	End of S wave and onset of T wave	Isoelectric	0.08	–

recorded as a separate wave in ECG because it merges with ventricular repolarization (QRS complex).

Duration

Normal duration of 'P' wave is 0.1 second.

Amplitude

Normal amplitude of 'P' wave is 0.1 to 0.12 mV.

Morphology

'P' wave is normally positive (upright) in leads I, II, aVF, V_4, V_5 and V_6. It is normally negative (inverted) in aVR. It is variable in the remaining leads, i.e. it may be positive, negative, biphasic or flat (Fig. 94.4).

Clinical Significance

Variation in the duration, amplitude and morphology of 'P' wave helps in the diagnosis of several cardiac problems such as:

1. *Right atrial hypertrophy:* 'P' wave is tall (more than 2.5 mm) in lead II. It is usually pointed
2. *Left atrial dilatation or hypertrophy:* It is tall and broad based or M shaped
3. *Atrial extrasystole:* Small and shapeless 'P' wave, followed by a small compensatory pause
4. *Hyperkalemia:* 'P' wave is absent or small
5. *Atrial fibrillation:* 'P' wave is absent
6. *Middle AV nodal rhythm:* 'P' wave is absent
7. *Sinoatrial block:* 'P' wave is inverted or absent
8. *Atrial paroxysmal tachycardia:* 'P' wave is inverted
9. *Lower AV nodal rhythm:* 'P' wave appears after QRS complex.

■ 'QRS' COMPLEX

'QRS' complex is also called the **initial ventricular complex.** 'Q' wave is a small negative wave. It is continued as the tall 'R' wave, which is a positive wave. 'R' wave is followed by a small negative wave, the 'S' wave.

Cause

'QRS' complex is due to **depolarization** of **ventricular musculature.** 'Q' wave is due to the depolarization of basal portion of interventricular septum. 'R' wave is due to the depolarization of apical portion of interventricular septum and apical portion of ventricular muscle. 'S' wave is due to the depolarization of basal portion of ventricular muscle near the atrioventricular ring.

Duration

Normal duration of 'QRS' complex is between 0.08 and 0.10 second.

Amplitude

Amplitude of 'Q' wave = 0.1 to 0.2 mV.
Amplitude of 'R' wave = 1 mV.
Amplitude of 'S' wave = 0.4 mV.

Morphology

'Q' wave is normally small with amplitude of 4 mm or less. It is less than 25% of amplitude of 'R' wave in leads I, II, aVL, V_5 and V_6. In the remaining leads, its amplitude is < 0.2 mm.

From chest leads V_1 to V_6, 'R' wave becomes gradually larger. It is smaller in V_6 than V_5. 'S' wave is large in V_1 and larger in V_2. It gradually becomes smaller from V_3 to V_6.

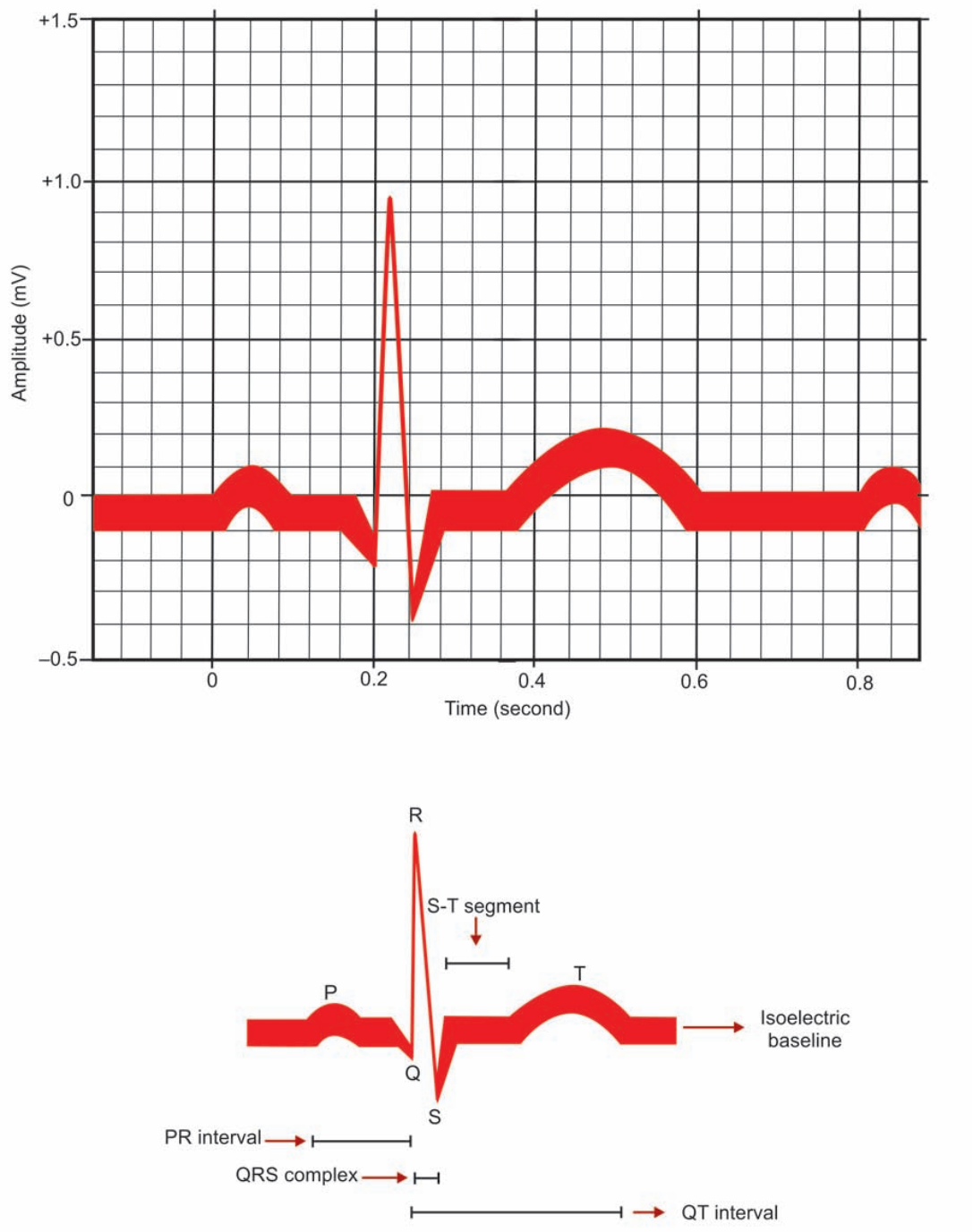


FIGURE 94.3: Waves of normal ECG

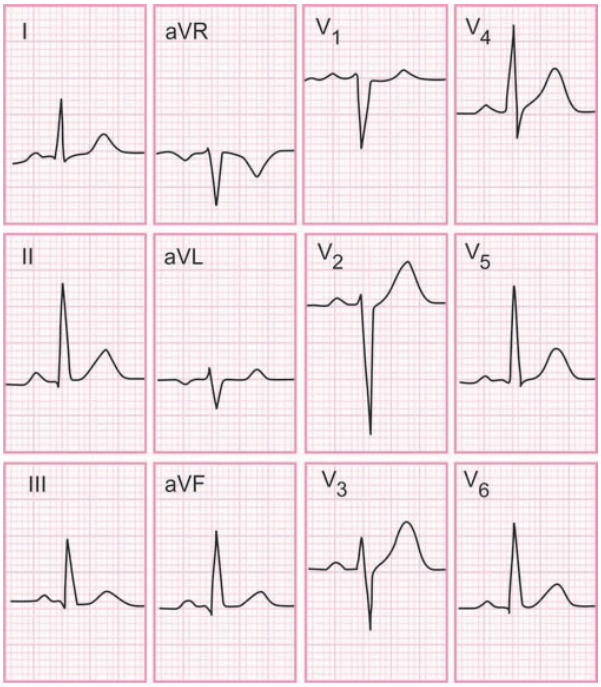


FIGURE 94.4: 12-lead ECG
(*Courtesy:* Dr Atul Ruthra)

Clinical Significance

Variation in the duration, amplitude and morphology of 'QRS' complex helps in the diagnosis of several cardiac problems such as:

1. *Bundle branch block:* QRS is prolonged or deformed
2. *Hyperkalemia:* QRS is prolonged.

■ 'T' WAVE

'T' wave is the **final ventricular complex** and is a positive wave.

Cause

'T' wave is due to the **repolarization** of **ventricular musculature.**

Duration

Normal duration of 'T' wave is 0.2 second.

Amplitude

Normal amplitude of 'T' wave is 0.3 mV.

Morphology

'T' wave is normally positive in leads I, II and V_5 and V_6. It is normally inverted in lead aVR. It is variable in the other leads, i.e. it is positive, negative or flat.

Clinical Significance

Variation in duration, amplitude and morphology of 'T' wave helps in the diagnosis of several cardiac problems such as:

1. *Acute myocardial ischemia:* Hyperacute 'T' wave develops. Hyperacute 'T' wave refers to a tall and broad-based 'T' wave, with slight asymmetry.
2. *Old age, hyperventilation, anxiety, myocardial infarction, left ventricular hypertrophy and pericarditis:* 'T' wave is small, flat or inverted
3. *Hypokalemia:* 'T' wave is small, flat or inverted
4. *Hyperkalemia:* 'T' wave is tall and tented.

■ 'U' WAVE

'U' wave is not always seen. It is also an insignificant wave in ECG. It is supposed to be due to **repolarization** of **papillary muscle.**

Clinical Significance

Appearance of 'U' wave in ECG indicates some clinical conditions such as:

1. *Hypercalcemia, thyrotoxicosis and hypokalemia:* 'U' wave appears. It is very prominent in hypokalemia.
2. *Myocardial ischemia:* Inverted 'U' wave appears.

■ INTERVALS AND SEGMENTS OF ECG

■ 'P-R' INTERVAL

'P-R' interval is the interval between the onset of 'P' wave and onset of 'Q' wave.

'P-R' interval signifies the atrial depolarization and conduction of impulses through AV node. It shows the duration of conduction of the impulses from the SA node to ventricles through atrial muscle and AV node.

'P' wave represents the atrial depolarization. Short **isoelectric** (zero voltage) period after the end of 'P' wave represents the time taken for the passage of depolarization within AV node.

Duration

Normal duration of 'P-R interval' is 0.18 second and varies between 0.12 and 0.2 second. If it is more than 0.2 second, it signifies the delay in the conduction of impulse from SA node to the ventricles. Usually, the delay occurs in the AV node. So it is called the **AV nodal delay.**

Clinical Significance

Variation in the duration of 'P-R' intervals helps in the diagnosis of several cardiac problems such as:

1. It is prolonged in bradycardia and first degree heart block

2. It is shortened in tachycardia, Wolf-Parkinson-White syndrome, Lown-Ganong-Levine syndrome, Duchenne muscular dystrophy and type II glycogen storage disease.

■ 'Q-T' INTERVAL

'Q-T' interval is the interval between the onset of 'Q' wave and the end of 'T' wave.

'Q-T' interval indicates the ventricular depolarization and ventricular repolarization, i.e. it signifies the electrical activity in ventricles.

Duration

Normal duration of Q-T interval is between 0.4 and 0.42 second.

Clinical Significance

1. 'Q-T' interval is prolonged in long 'Q-T' syndrome, myocardial infarction, myocarditis, hypocalcemia and hypothyroidism
2. 'Q-T' interval is shortened in short 'Q-T' syndrome and hypercalcemia.

■ 'S-T' SEGMENT

'S-T' segment is the time interval between the end of 'S' wave and the onset of 'T' wave. It is an isoelectric period.

J Point

The point where 'S-T' segment starts is called 'J' point. It is the junction between the QRS complex and 'S-T' segment.

Duration of 'S-T' Segment

Normal duration of 'S-T' segment is 0.08 second.

Clinical Significance

Variation in the duration of 'S-T' segment and its deviation from isoelectric base indicates the pathological conditions such as:

1. Elevation of 'S-T' segment occurs in anterior or inferior myocardial infarction, left bundle branch block and acute pericarditis. In athletes, 'S-T' segment is usually elevated
2. Depression of 'S-T' segment occurs in acute myocardial ischemia, posterior myocardial infarction, ventricular hypertrophy and hypokalemia
3. 'S-T' segment is prolonged in hypocalcemia
4. 'S-T' segment is shortened in hypercalcemia.

■ 'R-R' INTERVAL

'R-R' interval is the time interval between two consecutive 'R' waves.

Significance

'R-R' interval signifies the duration of one cardiac cycle.

Duration

Normal duration of 'R-R' interval is 0.8 second.

Significance of Measuring 'R-R' Interval

Measurement of 'R-R' interval helps to calculate:

1. Heart rate
2. Heart rate variability.

1. *Heart Rate*

Heart rate is calculated by measuring the number of 'R' waves per unit time.

Calculation of heart rate

Time is plotted horizontally (X-axis). On X-axis, interval between two thick lines is 0.2 sec (see above). Time duration for 30 thick lines is 6 seconds. Number of 'R' waves (QRS complexes) in 6 seconds (30 thick lines) is counted and multiplied by 10 to obtain heart rate. For the sake of convenience, the ECG paper has special time marking at every 3 seconds. So it is easy to find the time duration of 6 seconds.

2. *Heart Rate Variability*

Heart rate variability (HRV) refers to the beat-to-beat variations. Under resting conditions, the ECG of healthy individuals exhibits some periodic variation in 'R-R' intervals. This rhythmic phenomenon is known as **respiratory sinus arrhythmia** (RSA), since it fluctuates with the phases of respiration. 'R-R' interval decreases during inspiration and increases during expiration (Chapter 96).

Significance of Heart Rate Variability

HRV decreases in many clinical conditions like:

1. Cardiovascular dysfunctions such as hypertension
2. Diabetes mellitus
3. Psychiatric problems such as panic and anxiety.

Vector

Chapter 95

- INTRODUCTION
- INSTANTANEOUS MEAN VECTOR
- DEGREE OF INSTANTANEOUS MEAN VECTOR
  - DEGREE OF INSTANTANEOUS MEAN VECTOR AT DIFFERENT LIMB LEADS
- CALCULATED VECTOR OR MEAN QRS VECTOR
  - CALCULATION OF MEAN QRS VECTOR
- VECTORAL ANALYSIS
- VECTOR CARDIOGRAM

INTRODUCTION

Cardiac vector is the direction at which electrical potential generated in the heart travels at an instant. It is also called **cardiac axis.**

Vector is represented by an arrow. Arrowhead shows the **direction** of electrical potential. Length of the arrow represents the **amplitude** (magnitude or voltage) of the potential.

INSTANTANEOUS MEAN VECTOR

Current flows in all directions. Mean direction of flow of electrical potential at one instance is known as instantaneous mean vector or instantaneous summated vector (Fig. 95.1).

For example, when current flows through interventricular septum from the base of ventricles towards apex, the electrical potential generated by flow of current travels in different directions as follows:

1. Electrical potential travels downwards through the interventricular septum, towards the apical part, i.e. from depolarized part of septum towards non-depolarized (polarized) part of septum. This potential is strong.
2. Through the inner surface of ventricles, the potential travels upwards from apical part towards the base. Magnitude of this potential is very weak.

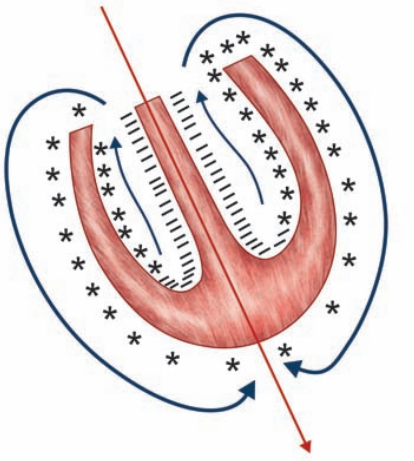


FIGURE 95.1: Instantaneous mean vector when current flows through interventricular septum of the heart

3. Through the outer surface of heart, the electrical potential travels downwards. It has a higher magnitude.

Though the potential travels in all directions in this instance, the potential flowing downwards (from base to apex of the heart) is much greater in magnitude than the potential flowing in other directions. Thus, the mean direction of flow of electrical potential in this instance is downwards. This downward vector is called

instantaneous mean vector or instantaneous summated vector at this instance.

■ DEGREE OF INSTANTANEOUS MEAN VECTOR

While recording electrocardiogram (ECG) in different limb leads, the degree of vector is altered. Direction of current flow is always from negative point towards the positive point. When the electrical potential flows in a horizontal plane from right side towards left side of the heart, the degree of vector is zero (Fig. 95.2).

■ DEGREE OF INSTANTANEOUS MEAN VECTOR AT DIFFERENT LIMB LEADS

Standard Limb Lead I (Right Arm and Left Arm)

In this instance, the electrical potential travels from right side (negative point) of the heart towards the left side (positive point) in the horizontal plane. So, the degree of vector is considered as zero.

Standard Limb Lead II (Right Arm and Left Leg)

Vector is from above downwards and slightly towards left, i.e. at 60°.

Standard Limb Lead III (Left Arm and Left Leg)

Here, vector is from above downwards and slightly towards right at 120°.

Lead Augmented Vector Right (aVR)

Vector is from below towards upper part of the heart and slightly towards right at 210°.

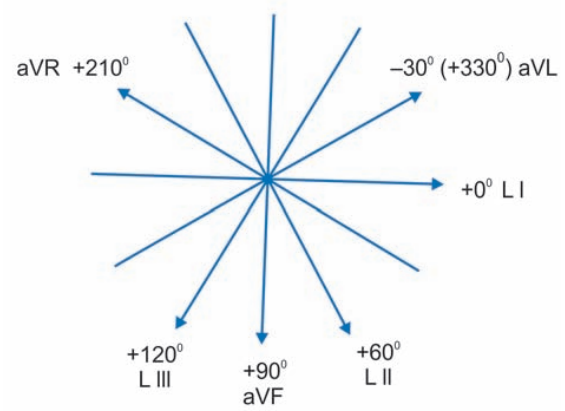


FIGURE 95.2: Degree of instantaneous vector at different leads

Lead Augmented Vector Front (aVF)

Vector is from above downwards at 90°.

Lead Augmented Vector Left (aVL)

In this, the vector is from below, towards upper part of the heart and slightly towards left, at –30° or at +330°.

■ CALCULATED VECTOR OR MEAN QRS VECTOR

Instantaneous mean vector cannot be determined by the recording of ECG. But, another vector can be calculated by measuring the amplitude of QRS complex from the ECG, recorded in standard limb leads. It is called the calculated vector or mean QRS vector.

It is also called the electrical axis of the heart or cardiac vector.

Calculated cardiac vector is useful in the diagnosis of heart diseases.

■ CALCULATION OF MEAN QRS VECTOR

Calculation of mean QRS vector depends upon the fact that if the amplitude of QRS complex is determined from ECG recorded at any two standard limb leads, the amplitude of QRS complex in the remaining lead can be known from the calculation.

Amplitude is measured in mm. For determining the amplitude of QRS complex, first the height of R wave is measured. From this value, height of negative wave Q or S (whichever is more) is deducted. The calculation is based on Einthoven triangle.

Einthoven Triangle

Refer previous Chapter for Einthoven triangle.

Steps for Calculation of Mean QRS Vector

1. An equilateral triangle is drawn on a plain paper. This triangle represents Einthoven triangle. Each side of this triangle represents one standard limb lead.
2. From the midpoint of each side, a perpendicular line is drawn towards the center. Meeting point of the perpendicular lines represents center of electrical activity in the heart (Fig. 95.3).
3. On each side of triangle, the amplitude of QRS complex is plotted from midpoint towards the positive point of the lead. For example, the amplitude of QRS complex in lead I is 10 mm and in lead II, it is 16 mm (Fig. 95.4).
4. In the triangle, upper side represents lead I and in this lead, the left is positive. So, a 10 mm line is drawn

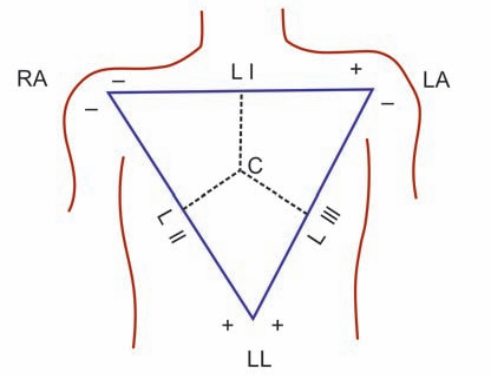


FIGURE 95.3: Einthoven triangle. C = Center of electrical activity, RA = Right arm, LA = Left arm, LL = Left leg, LI, LII and LIII = Standard limb leads.

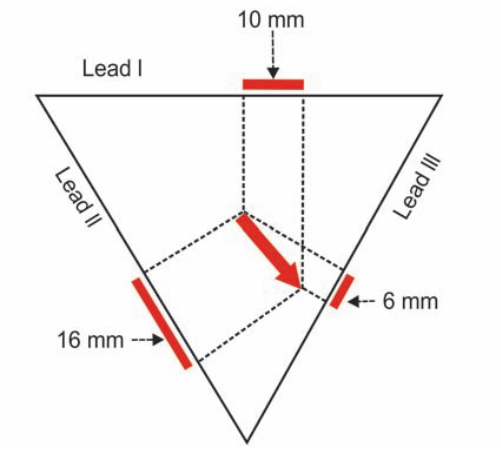


FIGURE 95.4: Calculation of cardiac vector. Arrow in the center indicates the cardiac vector.

on upper side from the midpoint, towards left (positive). This 10 mm distance along the axis of lead I is called **projected vector** for lead I.

5. In the same way, the projected vector for Lead II is drawn on the right side of the triangle
6. From the positive end of each projected vector another perpendicular line is drawn towards interior of the triangle
7. Now an arrow is drawn between center of electrical activity and the meeting point of perpendicular lines from positive end of projected vectors (Fig. 95.5).

   This arrow shows the vector. Arrowhead is drawn towards positive end, i.e. downwards.
8. Degree and the length of the arrow are measured. Degree denotes the direction of vector and length denotes the magnitude.

Amplitude of QRS Complex in Lead III

Amplitude (electrical potential) of QRS complex in lead III can be calculated by applying Einthoven law.

Einthoven law

Einthoven law states that potential differences between the bipolar leads measured simultaneously will, at any given moment, have the values II = I + III. That is, the potential of any wave or complex in lead II of ECG is equal to the sum of potentials in lead I and lead III. Einthoven law is the modification of Kirchhoff's law of voltage.

Kirchhoff's law of voltage

According to Kirchhoff law, the algebraic sum of voltage rise in a closed circuit is equal to the algebraic sum of voltage drops.

Application of Einthoven law in calculating QRS complex

By applying Einthoven law, amplitude (electrical potential) of QRS complex in one lead can be mathematically calculated, by summing up or subtracting the amplitude in other two leads, depending upon the potentials of these leads.

For example, amplitude of QRS in lead II = I + III and the amplitude of QRS in lead III = II – I. Thus, in this case of Einthoven triangle mentioned above, amplitude of QRS in lead I is 1 mV and lead II is 1.6 mV. Thus, the amplitude of QRS in lead III is 0.6 mV. It can also be measured from the triangle drawn to calculate the vector (Fig. 95.5).

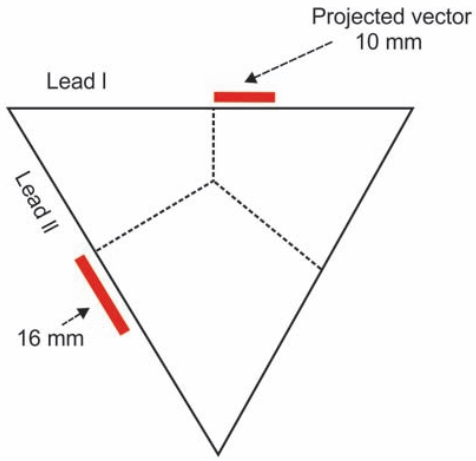


FIGURE 95.5: Determination of vector in Lead I and Lead II

■ VECTORAL ANALYSIS

Mean QRS vector (cardiac axis) in normal conditions is at about +59°. It varies between –30° and +110°.

When the axis deviates towards the left, i.e. in anti-clockwise direction, away from –30°, it is called left axis deviation. When the axis deviates towards the right (clockwise direction), away from +110°, it is known as right axis deviation.

Left axis deviation

Left axis deviation occurs in left ventricular hypertrophy, left bundle-branch block and posterior wall infarction.

Right axis deviation

Right axis deviation occurs due to right ventricular hypertrophy, right bundle-branch block and anterior wall infarction.

■ VECTOR CARDIOGRAM

From the recording of the electrocardiogram, only the calculated vector, i.e. cardiac axis is determined. Instantaneous mean vector cannot be determined by the electrocardiogram, but it can be determined by means of vector cardiogram.

Vector cardiogram is the simultaneous recording of electrical potential in different axis across the heart above, downward and sideward. It is obtained by using a cathode-ray oscilloscope. The technique is equal to connecting the tops of all instantaneous mean vectors in the series of 3 loops. It is done by means of a sophisticated electronic device along with oscilloscope.

Each loop of electronic connection is used to record different vector cardiogram called P vector cardiogram, QRS vector cardiogram and T vector cardiogram.

Arrhythmia

Chapter 96

- DEFINITION
- CLASSIFICATION
- NORMOTOPIC ARRHYTHMIA
  - SINUS ARRHYTHMIA
  - SINUS TACHYCARDIA
  - SINUS BRADYCARDIA
- ECTOPIC ARRHYTHMIA
  - HEART BLOCK
  - EXTRASYSTOLE
  - PAROXYSMAL TACHYCARDIA
  - ATRIAL FLUTTER
  - ATRIAL FIBRILLATION
  - VENTRICULAR FIBRILLATION
- ABNORMAL PACEMAKER
- ARTIFICIAL PACEMAKER
- CURRENT OF INJURY

DEFINITION

Arrhythmia refers to **irregular heartbeat** or disturbance in the rhythm of heart. In arrhythmia, heartbeat may be fast or slow or there may be an extra beat or a missed beat. It occurs in physiological and pathological conditions.

CLASSIFICATION

In arrhythmia, SA node may or may not be the pacemaker. If SA node is not the pacemaker, any other part of the heart such as atrial muscle, AV node and ventricular muscle becomes the pacemaker.

Accordingly, arrhythmia is classified into two types:

A. Normotopic arrhythmia
B. Ectopic arrhythmia.

NORMOTOPIC ARRHYTHMIA

Normotopic arrhythmia is the irregular heartbeat, in which SA node is the pacemaker.

Normotopic arrhythmia is of three types:

1. Sinus arrhythmia
2. Sinus tachycardia
3. Sinus bradycardia.

SINUS ARRHYTHMIA

Sinus arrhythmia is a normal rhythmical increase and decrease in heart rate, in relation to respiration. It is also called **respiratory sinus arrhythmia** (RSA). Normal sinus rhythm means the normal heartbeat with SA node as the pacemaker.

Normal heart rate is 72 per minute. However, under physiological conditions, in a normal healthy person, heart rate varies according to the phases of respiratory cycle. Heart rate increases during inspiration and decreases during expiration.

ECG Changes

ECG is normal during sinus arrhythmia. Only the duration of R-R interval varies rhythmically according to phases

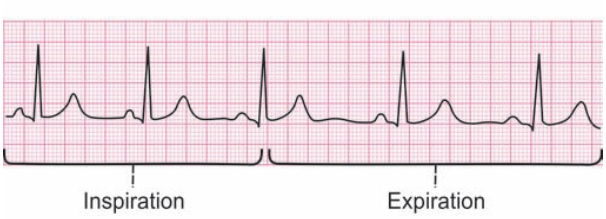


FIGURE 96.1: ECG in sinus arrhythmia. Normal P-QRS-T. R-R interval is shortened during inspiration and prolonged during expiration (*Courtesy:* Dr Atul Ruthra).

of respiration (Fig. 96.1). It is shortened during inspiration and prolonged during expiration (Chapter 94).

Cause

Sinus arrhythmia is due to fluctuation in the discharge of impulses from SA node (Fig. 96.2). During inspiration, the lungs are inflated and the intrathoracic pressure decreases. This increases the venous return. Inflation of lungs stimulates the stretch receptors of lungs, which send impulses to vasodilator area (cardioinhibitory center) through afferent fibers of vagus. It leads to reflex inhibition of vasodilator area and reduction in vagal tone. Because of these two factors, heart rate increases. Simultaneously, increased venous return initiates **Bainbridge reflex** that causes increase in heart rate (Chapter 101).

During expiration, the lungs are deflated and intrathoracic pressure increases. This decreases the venous return. During deflation of lungs, the stretch receptors are not stimulated and vasodilator area is not inhibited. So, vagal tone increases, resulting in decreased heart rate. Simultaneously, decreased venous return abolishes Bainbridge reflex. It also decreases the heart rate.

■ SINUS TACHYCARDIA

Sinus tachycardia is the increase in discharge of impulses from SA node, resulting in increase in heart rate. Discharge of impulses from SA node is very rapid and the heart rate increases up to 100/minute and sometimes up to 150/minute.

ECG Changes

ECG is normal in sinus tachycardia, except for short R-R intervals because of increased heart rate (Fig. 96.3).

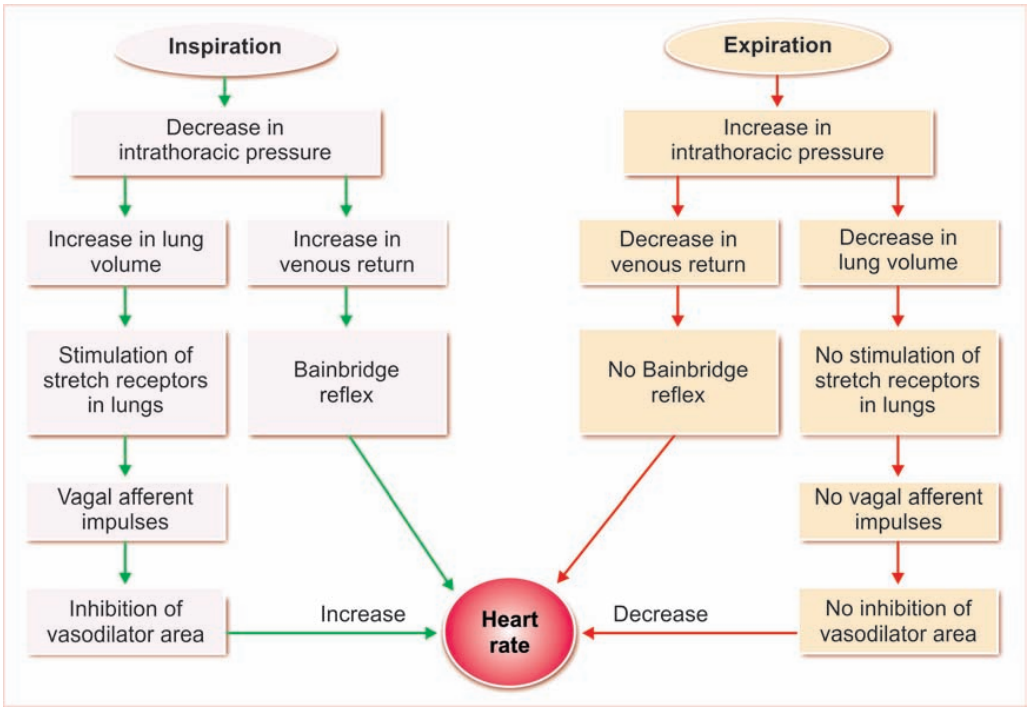


FIGURE 96.2: Sinus arrhythmia

FIGURE 96.3: ECG in sinus tachycardia. Normal P-QRS-T. R-R interval is shortened. Heart rate is more than 100/min (*Courtesy:* Dr Atul Ruthra).

Conditions when Sinus Tachycardia Occurs

Sinus tachycardia occurs in physiological as well as pathological conditions.

Physiological conditions when tachycardia occurs

1. Exercise
2. Emotion
3. High altitude
4. Pregnancy.

Pathological conditions when tachycardia occurs

1. Fever
2. Anemia
3. Hyperthyroidism
4. Hypersecretion of catecholamines
5. Cardiomyopathy
6. Valvular heart disease
7. Hemorrhagic shock.

Features of Sinus Tachycardia

1. Palpitations (sensation of feeling the heartbeat)
2. Dizziness
3. Fainting
4. Shortness of breath
5. Chest discomfort (angina).

■ SINUS BRADYCARDIA

Sinus bradycardia is the reduction in discharge of impulses from SA node resulting in decrease in heart rate. Heart rate is less than 60/minute.

ECG Changes

ECG shows prolonged waves and prolonged R-R interval (Fig. 96.4).

Conditions when Sinus Bradycardia Occurs

Sinus bradycardia occurs in both physiological and pathological conditions. It occurs during sleep. It is common in athletes due to the cardiovascular reflexes, in response to increased force of contraction of heart.

Physiological conditions when sinus bradycardia occurs

1. Sleep
2. Athletic heart.

Pathological conditions when sinus bradycardia occurs

1. Disease of SA node
2. Hypothermia
3. Hypothyroidism
4. Heart attack
5. Congenital heart disease
6. Degenerative process of aging
7. Obstructive jaundice
8. Increased intracranial pressure
9. Use of certain drugs like beta blockers, channel blockers, digitalis and other **antiarrhythmic drugs**
10. Atherosclerosis. Bradycardia due to **atherosclerosis** of carotid artery, at the region of carotid sinus is called **carotid sinus syndrome.**

Features of Sinus Bradycardia

1. Sick sinus syndrome
2. Fatigue
3. Weakness
4. Shortness of breath
5. Lack of concentration
6. Difficulty in exercising.

Sick sinus syndrome

Sick sinus syndrome is the common feature of sinus bradycardia. It is the condition characterized by dizziness and unconsciousness.

■ ECTOPIC ARRHYTHMIA

Ectopic arrhythmia is the abnormal heartbeat, in which one of the structures of heart other than SA node becomes the pacemaker. Impulses produced by these structures are called **ectopic foci.**

FIGURE 96.4: ECG in sinus bradycardia. Normal P-QRS-T. R-R interval is prolonged. Heart rate is less than 60/min. (*Courtesy:* Dr Atul Ruthra).

Subtypes of Ectopic Arrhythmia

Ectopic arrhythmia is further divided into two subtypes:

1. Homotopic arrhythmia, in which the impulses for heartbeat arise from any part of conductive system
2. Heterotopic arrhythmia, in which the impulses arise from the musculature of heart other than conductive system.

Different Ectopic Arrhythmia

1. Heart block
2. Extrasystole
3. Paroxysmal tachycardia
4. Atrial flutter
5. Atrial fibrillation
6. Ventricular fibrillation.

■ HEART BLOCK

Heart block is the blockage of impulses generated by SA node in the conductive system. Because of the blockage, the impulses cannot reach the cardiac musculature, resulting in ectopic arrhythmia. Based on the area affected, the heart block is classified into two types (Fig. 96.5):

1. Sinoatrial block
2. Atrioventricular block.

Sinoatrial Block – AV Nodal Rhythm

Sinoatrial block is the failure of impulse transmission from SA node to AV node. It is also called **sinus block.** During sinoatrial block, heart stops beating. Immediately, AV node takes over the pacemaker function and produces the impulses. This leads to AV nodal (atrioventricular) rhythm.

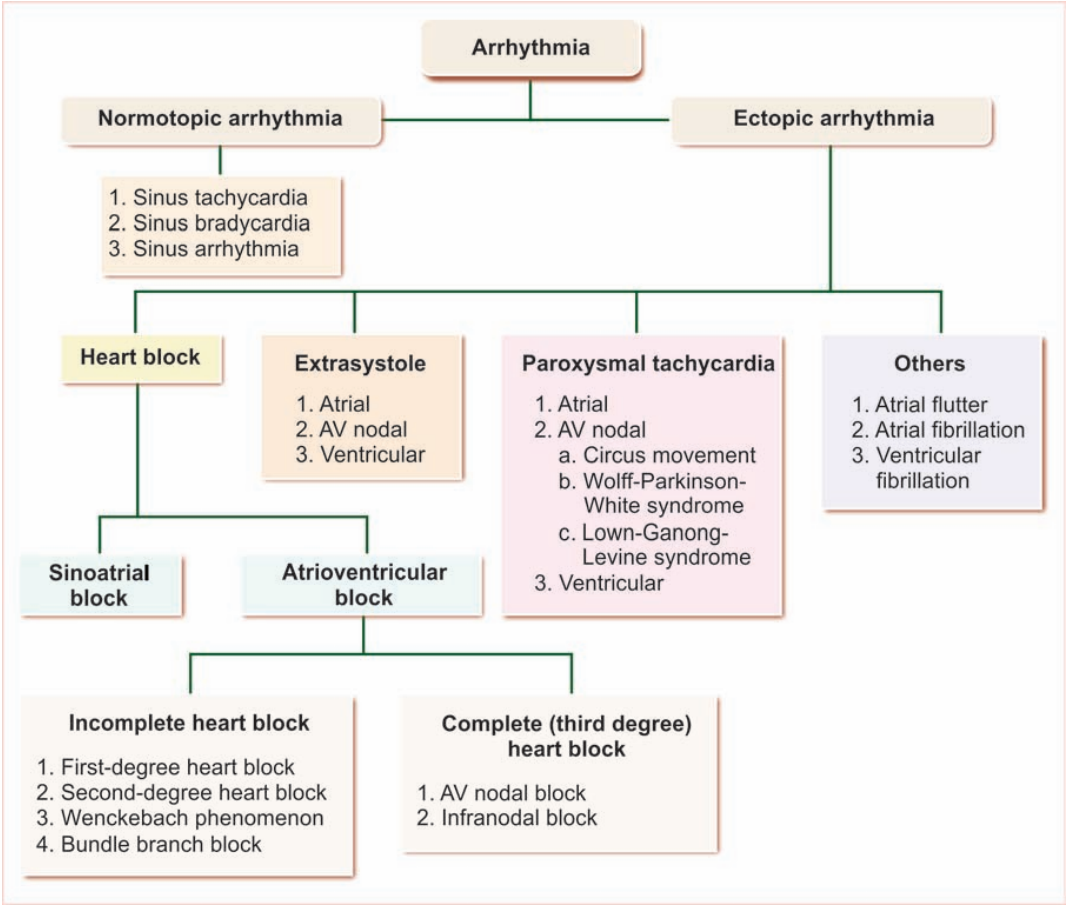


FIGURE 96.5: Classification of arrhythmia. AV = Atrioventricular.

Sinoatrial block is due to the defect in internodal fibers and it occurs suddenly. Initially, the heart stops for a while. Then after few seconds, the AV node becomes the pacemaker and the heart starts beating with decreased rate of 40 to 60/minute.

Impulses may be discharged from any part of AV node, viz.

1. In upper nodal rhythm, the impulses are discharged from the upper part of AV node. In this rhythm, the P wave of ECG is inverted. QRS complex and T wave are normal
2. In middle nodal rhythm, the impulses are by the middle part of AV node. Here, all the chambers of the heart contract simultaneously. P wave of ECG is absent as it merges with QRS complex
3. In lower nodal rhythm, the impulses are produced by the lower part of AV node. In this condition, ventricular contraction occurs prior to atrial contraction as the impulses reach the ventricles prior to the atria. In ECG, QRS complex appears prior to P wave and R-P interval is obtained instead of P-R interval. It is called **reversed heart block.**

Atrioventricular Block

Atrioventricular block is the heart block in which the impulses are not transmitted from atria (from AV node) to ventricles because of defective conductive system.

Atrioventricular block is of two categories:

1. Incomplete heart block
2. Complete heart block.

1. *Incomplete Heart Block*

Incomplete heart block is the condition in which the transmission of impulses from atria to ventricles is slowed down and not blocked completely. Impulses reach ventricles late.

Incomplete heart block is of four types:

i. First degree heart block
ii. Second degree heart block
iii. Wenckebach phenomenon
iv. Bundle branch block.

i. *First degree heart block*

First degree heart block is the heart block in which the conduction of impulses through AV node is very slow, i.e. the AV nodal delay is longer. It is also called **delayed conduction.** In ECG, the P-R interval is very much prolonged and is more than 0.2 second.

First degree heart block is common in young adults and trained athletes. It is also caused by rheumatic fever and some drugs. It does not produce any symptom.

ii. *Second degree heart block*

Second degree heart block is the type of heart block in which some of the impulses produced by SA node fail to reach the ventricles. It is also called the **partial heart block.** When some of the impulses from SA node fail to reach the ventricles, one ventricular contraction occurs for every 2, 3 or 4 atrial contractions, i.e. 2 : 1, 3 : 1 or 4 : 1. In ECG, the ventricular complex (QRST) is missing accordingly.

During frequent development of second degree heart block, bradycardia occurs.

iii. *Wenckebach phenomenon or syndrome*

Wenckebach phenomenon is a type of heart block characterized by progressive increase in AV nodal delay, resulting in missing of one beat. Afterwards, the conduction of impulse is normal or slightly delayed. In ECG, the progressive lengthening of P-R interval is noticed till QRST complex disappears.

iv. *Bundle branch block*

Bundle branch block (BBB) is the heart block that occurs during dysfunction of right or left branch of bundle of His. During this type of block, the impulse from atria reaches unaffected ventricle first. Then, from here, the impulse travels to the affected side. So, ECG shows normal ventricular rate, but the QRS complex is prolonged or deformed.

2. *Complete Heart Block (Third degree heart block)*

Complete heart block is the condition in which the impulses produced by SA node cannot reach the ventricles. It is also called **complete atrioventricular block** or third degree heart block. Because of this, the ventricles beat in their own rhythm, independent of atrial beat. It is called **idioventricular rhythm.**

Complete heart block occurs due to any one of the following causes:

i. Disease of AV node, which leads to AV nodal block
ii. Defective conductive system below the level of AV node, causing infranodal block.

i. *AV nodal block*

In this type of block, a part of AV node is defective and the unaffected part becomes the pacemaker. Rhythmicity of AV node is about 45 to 60/minute.

ii. *Infranodal block*

Infranodal block is the heart block in which the impulses from SA node are blocked in the branches of bundle of His (below the level of AV node). In this condition, the

distal part of the conductive system (i.e. the Purkinje fibers) becomes the pacemaker. The rhythmicity of Purkinje fibers is about 35/minute. Sometimes, a part of ventricular musculature becomes the pacemaker and the ventricular rate in such conditions is about 20/minute.

Third degree heart block is the serious one since it decreases the pumping action of the heart. Very often, it results in Stokes-Adams syndrome. It may also cause heart failure.

Stokes-Adams syndrome

Stokes-Adams syndrome is the sudden attack of dizziness and unconsciousness caused by heart block. It may be accompanied by convulsions also. In many patients suffering from heart block, the complete heart block occurs intermittently. When the block occurs, the ventricles stop beating immediately. **Ectopic pacemaker** (AV node, Purkinje fiber or ventricular muscle) starts functioning only after 5 to 30 seconds.

During this time, the blood circulation is affected because of lack of ventricular output. Brain cannot withstand the stoppage of blood supply and oxygen supply even for 5 seconds. Before the onset of discharge from ectopic pacemaker, dizziness and fainting occurs. If the discharge of impulses from ectopic pacemaker is delayed beyond 30 seconds, death occurs.

■ EXTRASYSTOLE

Extrasystole and Compensatory Pause

Extrasystole is the premature contraction of the heart before its normal contraction. It is caused by an ectopic focus (discharge of an impulse from any part of the heart other than the SA node). The ectopic focus produces an extra beat of the heart that is always followed by a compensatory pause. Compensatory pause is the period during which the heart stops in relaxed state.

Cause for the compensatory pause

In the cardiac muscle, absolute refractory period extends throughout contraction period. When the heart is in extrasystole (because of ectopic focus), an impulse is discharged from natural pacemaker, SA node. As this natural impulse reaches the myocardium during the contraction period of extrasystole, the myocardium does not give response, because it is refractory now. For the next beat, the heart has to wait till the discharge of next natural impulse from SA node. During this time, the heart stops in diastole. It is the cause for compensatory pause (Chapter 90).

Parts of the heart which give origin for ectopic foci are AV node, bundle of His, atrial musculature and ventricular musculature.

Accordingly, extrasystole is divided into three types:

1. Atrial extrasystole
2. Nodal extrasystole
3. Ventricular extrasystole.

1. *Atrial Extrasystole*

Atrial extrasystole is the premature contraction produced by a stimulus arising from atrial muscle. In this condition, an extra P wave appears immediately after the regular T wave. P wave is small and shapeless. The P-R interval of this beat is short.

2. *Nodal Extrasystole*

Nodal extrasystole is caused by stimulus arising from AV node. P wave is merged with QRS complex and all the chambers of the heart contract together.

3. *Ventricular Extrasystole*

Ventricular extrasystole is the extrasystole that is caused by stimulus from ventricular muscle. In this condition, an extra QRS complex follows the regular T wave. This QRS complex is prolonged as the impulse is conducted through ventricular muscle and not through the conductive system. This QRS complex also has a high voltage. T wave of this beat is inverted.

Conditions when Extrasystole Occurs

Extrasystole is associated with organic diseases of the heart. Particularly, any ischemic area of ventricular musculature can produce an ectopic focus.

Other conditions which produce extrasystole:

i. Emotions
ii. Severe exhaustion
iii. Excessive ingestion of coffee or alcohol
iv. Excessive smoking
v. Hyperthyroidism
vi. Reflexes elicited from abnormal viscera.

■ PAROXYSMAL TACHYCARDIA

Paroxysmal tachycardia is the sudden attack of increased heart rate due to ectopic foci arising from atria, AV node or ventricle. It is also called **Bouveret-Hoffmann syndrome.**

Increase in heart rate due to ectopic foci arising from either atria or AV node is called **supraventricular**

tachycardia (SVT). It differs from ventricular tachycardia, which does not depend upon atria or AV node. The attack lasts for a period of few seconds to few hours. It also stops suddenly. After the attack, heart functions normally. Symptoms include palpitations, chest pain, rapid breathing and dizziness.

Paroxysmal tachycardia is of three types:

1. Atrial paroxysmal tachycardia
2. AV nodal paroxysmal tachycardia
3. Ventricular paroxysmal tachycardia.

1. *Atrial Paroxysmal Tachycardia*

Atrial paroxysmal tachycardia is the sudden increase in heart rate caused by ectopic impulses discharged from atrial musculature. Heart rate is 150 to 220/minute. P wave in ECG is inverted, with normal QRST.

2. *AV Nodal Paroxysmal Tachycardia – Bundle of Kent*

AV nodal paroxysmal tachycardia is the sudden increase in heart rate caused by ectopic foci arising from AV node due to a temporary block in the conductive system. It also involves circus movement. This type of tachycardia is very common in some healthy persons who have got an additional conductive system. This system is formed by some abnormal junctional tissues constituting a structure called bundle of Kent. Bundle of Kent connects the atria and ventricles directly, so the conduction is very rapid than through the regular conductive system.

Circus movement – Re-entry and atrial echo beat

Circus movement is defined as circuitous propagation of impulses around a structural or functional obstruction, resulting in re-entry of the impulse and **re-excitation** of heart. When there is a sudden and temporary block in normal conductive system, the impulses from SA node reach the ventricle through bundle of Kent. By this time, the blockage in normal conductive system disappears. Now, the impulse, which passes through bundle of Kent, after exciting the ventricular muscle, travels in the opposite direction through the normal conductive system and finally, it re-enters the AV node. Re-entered impulse activates the AV node and depolarizes the atria, resulting in atrial contraction. It is called **atrial echo beat.**

Re-entered nodal impulse simultaneously spreads to ventricle through normal conductive system, completing the circus movement. This circus movement is repeated producing tachycardia called AV nodal paroxysmal tachycardia. ECG shows normal QRST complex. But P wave is mostly absent.

Wolff-Parkinson-White syndrome

Wolff-Parkinson-White syndrome is the condition characterized by repeated attacks of AV nodal paroxysmal tachycardia in persons with bundle of Kent. ECG shows short P-R interval with normal QRS complex and T wave.

Lown-Ganong-Levin syndrome

Lown-Ganong-Levin syndrome is another condition characterized by AV nodal paroxysmal tachycardia. This occurs in persons who have got another type of abnormal conductive fibers like bundle of Kent. These fibers also connect atria and distal part of conductive system directly bypassing the AV node. So the impulse from SA node reaches ventricle through the abnormal conductive fibers. After exciting the ventricular muscle, the impulse travels in opposite direction through normal conductive system and finally, it re-enters the AV node. The re-entered impulse activates the AV node causing atrial contraction. ECG shows short P-R interval with normal QRS complex and T wave.

3. *Ventricular Paroxysmal Tachycardia*

Ventricular paroxysmal tachycardia is the sudden increase in heart rate caused by ectopic foci arising from ventricular musculature. Sometimes, a part of ventricular muscle, particularly an ischemic area is excited abnormally, followed by a series of extrasystole. This condition is dangerous as the circus movement is developed within ventricular muscle. This circus movement leads to ventricular fibrillation, which is fatal.

■ ATRIAL FLUTTER

Atrial flutter is an arrhythmia characterized by rapid ineffective atrial contractions, caused by ectopic foci originating from atrial musculature. It is often associated with atrial paroxysmal tachycardia. Both the atria beat rapidly like the wings of a bird, hence the name atrial flutter.

Atrial rate is about 250 to 350/minute. Maximum number of impulses conducted by AV node is about 230 to 240 /minute. So, during atrial flutter, the second degree of heart block occurs. The ratio between atrial beats and ventricular beats is 2 : 1 or sometimes 3 : 1.

Atrial flutter is common in patients suffering from cardiovascular diseases such as hypertension and coronary artery disease. Initially, it is marked by palpitations that are unnoticed. However, prolonged atrial flutter may lead to atrial fibrillation or heart failure.

■ ATRIAL FIBRILLATION

Atrial fibrillation is the type of arrhythmia characterized by rapid and irregular atrial contractions at the rate of 300 to 400 beats/minute. It is mostly due to circus movement of impulses within atrial musculature. P wave is absent in ECG.

Atrial fibrillation is common in old people and patients with heart diseases. Though it is not life-threatening, it may cause complications. If it continues for long time, it may cause blood clot and blockage of blood flow to vital organs.

■ VENTRICULAR FIBRILLATION

Ventricular fibrillation is the dangerous cardiac arrhythmia, characterized by rapid and irregular twitching of ventricles. Ventricles beat very rapidly and irregularly due to the circus movement of impulses within ventricular muscle. The rate reaches 400 to 500/minute. This is triggered by ventricular extrasystole. This type of arrhythmia is serious as it leads to death, since the ventricles cannot pump blood.

Ventricular fibrillation is very common during electric shock and during ischemia of conductive system. It also occurs in other conditions like coronary occlusion, chloroform anesthesia, cyclopropane anesthesia, trauma of heart and disturbances of heart (due to improper handling) during cardiac surgery.

■ ABNORMAL PACEMAKER

Abnormal pacemaker is the part of the heart other than SA node that becomes the pacemaker and discharges ectopic foci. Various types of arrhythmia develop when an abnormal pacemaker is activated. These arrhythmias are already described in this Chapter.

Common abnormal pacemakers:

1. Atrioventricular node
2. Atrial musculature
3. Ventricular musculature.

1. *AV Node as Pacemaker*

When AV node becomes the pacemaker, the following arrhythmias occur:

i. AV nodal rhythm
ii. AV nodal extrasystole
iii. AV nodal paroxysmal tachycardia.

2. *Atrial Musculature as Pacemaker*

Following arrhythmias occur if atrial musculature becomes the pacemaker:

i. Atrial extrasystole
ii. Atrial paroxysmal tachycardia
iii. Wolff-Parkinson-White syndrome
iv. Lown-Ganong-Levine syndrome
v. Atrial flutter
vi. Atrial fibrillation.

3. *Ventricular Musculature as Pacemaker*

If ventricular muscle becomes the pacemaker, following arrhythmias are developed:

i. Ventricular extrasystole
ii. Ventricular paroxysmal tachycardia
iii. Ventricular fibrillation.

■ ARTIFICIAL PACEMAKER

Artificial pacemaker is a small **electronic device** that is surgically implanted to regulate abnormal heartbeat. It contains a battery powered **pulse generator,** that produces electrical impulses capable of stimulating the heart. This pacemaker is implanted under the skin over the chest of the patient. Pulses generated by this device are transmitted to the heart through electrodes. Electrodes connected to the device are inserted and passed through a vein and positioned in the heart chambers. The device has a **lithium battery** that may last for 10 to 15 years. The outer casing of the pacemaker is usually made of titanium, which is rarely rejected by body's immune system.

Pulse generator of the pacemaker has multiple functions. It is programmed to cope up with the needs of the individual patient.

■ CURRENT OF INJURY

Current of injury means flow of current from an injured region of heart to the unaffected part. When ischemia occurs in any part of the ventricular musculature due to coronary occlusion, that part of ventricle becomes depolarized either partially or completely and the repolarization does not occur. It causes flow of current from affected (depolarized) part to unaffected part of the ventricular muscle.

Current of injury in myocardial infarction affects the ECG pattern and cardiac vector. In ECG, the J point and ST segments are displaced (Chapter 94). Deviation of cardiac axis is also common during the current of injury.

Cardiac Axis

In the infarction of anterior wall of the ventricle, the cardiac axis (vector) is deviated to right up to +150° due to current of injury and in the posterior wall infarction, there is left axis deviation up to –95°.

Effect of Changes in Electrolyte Concentration on Heart

Chapter 97

- INTRODUCTION
- EFFECT OF CHANGES IN SODIUM ION CONCENTRATION
- EFFECT OF CHANGES IN POTASSIUM ION CONCENTRATION
  - EFFECT OF HYPERKALEMIA
  - EFFECT OF HYPOKALEMIA
- EFFECT OF CHANGES IN CALCIUM ION CONCENTRATION
  - EFFECT OF HYPERCALCEMIA
  - EFFECT OF HYPOCALCEMIA
- EXPERIMENTAL EVIDENCES

INTRODUCTION

Distribution of electrolytes in extracellular fluid and intracellular fluid is responsible for the electrical activity of the tissues including myocardium. Thus, any change in the concentration of any electrolyte will definitely alter the electrical activity of cardiac muscle.

EFFECT OF CHANGES IN SODIUM ION CONCENTRATION

Normal sodium ion concentration in blood is 135 to 145 mEq/L. Change in concentration of sodium ion does not alter the electrical activity of heart severely. Only the low level of sodium ion in body fluids reduces the electrical activity of cardiac muscle and electrocardiogram (ECG) shows low-voltage waves.

Changes in the concentration of potassium and calcium ions have significant effects on heart.

EFFECT OF CHANGES IN POTASSIUM ION CONCENTRATION

Normal potassium ion concentration in blood is about 3.5 to 5 mEq/L. Changes in ECG appear when the potassium level increases to 6 mEq/L (hyperkalemia) or when it decreases to 2 mEq/L (hypokalemia).

EFFECT OF HYPERKALEMIA

Hyperkalemia decreases:

1. Resting membrane potential, leading to hyperpolarization
2. Excitability of the muscle.

Effects of hyperkalemia on the excitability of cardiac muscle, depend upon the severity of hyperkalemia.

Changes in ECG When Potassium Level Increases to 6 or 7 mEq/L

T wave is tall and tented. P-R interval and QRS complex are normal.

Changes in ECG When Potassium Level Increases to 8 mEq/L

P-R interval and the duration of QRS complex are prolonged because, hyperkalemia decreases the rate of conduction. P wave may be small.

Changes in ECG When Potassium Level Increases beyond 9 mEq/L

Severe hyperkalemia makes the atrial muscle unexcitable. So, P wave is absent in ECG. QRS complex merges with T wave. This condition is fatal because, it

leads to ventricular fibrillation or stoppage of heart in diastole, due to the lack of excitability.

■ EFFECT OF HYPOKALEMIA

Hypokalemia decreases the sensitivity of heart muscle.

Changes in ECG When Potassium Level Falls to 2 mEq/L

1. S-T segment is depressed
2. T wave is small, flat or inverted
3. U wave appears. Sometimes, the U wave merges with T wave. Because of this, the Q-T interval is mistaken for being prolonged.

Changes in ECG When Potassium Level Falls below 2 mEq/L

1. Depression of S-T segment below the isoelectric baseline
2. Inversion of T wave
3. Appearance of prominent U wave
4. Prolongation of P-R interval.

■ EFFECT OF CHANGES IN CALCIUM ION CONCENTRATION

Normal concentration of calcium ion in blood is 9 to 11 mg/dL (4.5 to 5.5 mEq/L). Mostly, hypocalcemia affects the heart, rather than hypercalcemia.

■ EFFECT OF HYPERCALCEMIA

Hypercalcemia is the elevation in blood calcium level. It increases the excitability and contractility of the heart muscle. In clinical conditions, the effect of hypercalcemia is very rare.

Changes in ECG

1. Shortening of duration of S-T segment
2. Shortening of QT interval
3. Appearance of U wave.

Calcium Rigor

Stoppage of the heart in systole, due to hypercalcemia is called the calcium rigor. It can be demonstrated in experimental animals by infusing large quantity of calcium. Calcium rigor is a reversible phenomenon and the heart starts functioning normally, when the calcium ions are washed.

■ EFFECT OF HYPOCALCEMIA

Hypocalcemia is the reduction in blood calcium level. It reduces the excitability of the cardiac muscle.

Changes in ECG

1. Prolongation of S-T segment
2. Prolongation of Q-T interval
3. Appearance of a prominent U wave.

■ EXPERIMENTAL EVIDENCES

Effects of ions on heart are demonstrated experimentally by perfusion of heart from animals such as frog and rabbit.

Cardiac Output

Chapter 98

- INTRODUCTION
- DEFINITIONS AND NORMAL VALUES
  - STROKE VOLUME
  - MINUTE VOLUME
  - CARDIAC INDEX
- EJECTION FRACTION
- CARDIAC RESERVE
- VARIATIONS IN CARDIAC OUTPUT
  - PHYSIOLOGICAL VARIATIONS
  - PATHOLOGICAL VARIATIONS
- DISTRIBUTION OF CARDIAC OUTPUT
- FACTORS MAINTAINING CARDIAC OUTPUT
  - VENOUS RETURN
  - FORCE OF CONTRACTION
  - HEART RATE
  - PERIPHERAL RESISTANCE
- MEASUREMENT OF CARDIAC OUTPUT
  - DIRECT METHODS
  - INDIRECT METHODS
- CARDIAC CATHETERIZATION
  - DEFINITION
  - CONDITIONS WHEN CARDIAC CATHETERIZATION IS PERFORMED
  - PROCEDURE
  - USES OF CARDIAC CATHETERIZATION

INTRODUCTION

Cardiac output is the amount of blood pumped from each ventricle. Usually, it refers to left ventricular output through aorta. Cardiac output is the most important factor in cardiovascular system, because rate of blood flow through different parts of the body depends upon cardiac output.

DEFINITIONS AND NORMAL VALUES

Usually, cardiac output is expressed in three ways:

1. Stroke volume
2. Minute volume
3. Cardiac index.

However, in routine clinical practice, cardiac output refers to minute volume.

STROKE VOLUME

Stroke volume is the amount of blood pumped out by each ventricle during each beat.

Normal value: 70 mL (60 to 80 mL) when the heart rate is normal (72/minute).

MINUTE VOLUME

Minute volume is the amount of blood pumped out by each ventricle in one minute. It is the product of stroke volume and heart rate:

Minute volume = Stroke volume × Heart rate

Normal value: 5 L/ventricle/minute.

CARDIAC INDEX

Cardiac index is the minute volume expressed in relation to square meter of body surface area. It is defined as the amount of blood pumped out per ventricle/minute/square meter of the body surface area.

Normal value: 2.8 ± 0.3 L/square meter of body surface area/minute (in an adult with average body surface area of 1.734 square meter and normal minute volume of 5 L/minute).

EJECTION FRACTION

Ejection fraction is the fraction of end diastolic volume that is ejected out by each ventricle. Normal ejection fraction is 60% to 65%. Refer Chapter 91 for details.

CARDIAC RESERVE

Cardiac reserve is the maximum amount of blood that can be pumped out by heart above the normal value. Cardiac reserve plays an important role in increasing the cardiac output during the conditions like exercise. It is essential to withstand the stress of exercise.

Cardiac reserve is usually expressed in percentage. In a normal young healthy adult, the cardiac reserve is 300% to 400%. In old age, it is about 200% to 250%. It increases to 500% to 600% in athletes. In cardiac diseases, the cardiac reserve is minimum or nil.

VARIATIONS IN CARDIAC OUTPUT

PHYSIOLOGICAL VARIATIONS

1. *Age:* In children, cardiac output is less because of less blood volume. Cardiac index is more than that in adults because of less body surface area.
2. *Sex:* In females, cardiac output is less than in males because of less blood volume. Cardiac index is more than in males, because of less body surface area.
3. *Body build:* Greater the body build, more is the cardiac output.
4. *Diurnal variation:* Cardiac output is low in early morning and increases in day time. It depends upon the basal conditions of the individuals.
5. *Environmental temperature:* Moderate change in temperature does not affect cardiac output. Increase in temperature above 30°C raises cardiac output.
6. *Emotional conditions:* Anxiety, apprehension and excitement increases cardiac output about 50% to 100% through the release of catecholamines, which increase the heart rate and force of contraction.
7. *After meals:* During the first one hour after taking meals, cardiac output increases.
8. *Exercise:* Cardiac output increases during exercise because of increase in heart rate and force of contraction.
9. *High altitude:* In high altitude, the cardiac output increases because of increase in secretion of adrenaline. Adrenaline secretion is stimulated by hypoxia (lack of oxygen).
10. *Posture:* While changing from recumbent to upright position, the cardiac output decreases.
11. *Pregnancy:* During the later months of pregnancy, cardiac output increases by 40%.
12. *Sleep:* Cardiac output is slightly decreased or it is unaltered during sleep.

PATHOLOGICAL VARIATIONS

Increase in Cardiac Output

Cardiac output increases in the following conditions:

1. *Fever:* Due to increased oxidative processes
2. *Anemia:* Due to hypoxia
3. *Hyperthyroidism:* Due to increased basal metabolic rate.

Decrease in Cardiac Output

Cardiac output decreases in the following conditions:

1. *Hypothyroidism:* Due to decreased basal metabolic rate
2. *Atrial fibrillation:* Because of incomplete filling of ventricles
3. *Incomplete heart block with coronary sclerosis or myocardial degeneration:* Due to defective pumping action of the heart
4. *Congestive cardiac failure:* Because of weak contractions of heart
5. *Shock:* Due to poor pumping and circulation
6. *Hemorrhage:* Because of decreased blood volume.

DISTRIBUTION OF CARDIAC OUTPUT

The whole amount of blood pumped out by the right ventricle goes to lungs. But, the blood pumped by the left ventricle is distributed to different parts of the body.

Fraction of cardiac output distributed to a particular region or organ depends upon the metabolic activities of that region or organ.

Distribution of Blood Pumped out of Left Ventricle

Distribution of blood pumped out of left ventricle to different organs and the percentage of cardiac output are given in Table. 98.1. Heart, which pumps the blood to all other organs, receives the least amount of blood. Liver receives maximum amount of blood.

■ FACTORS MAINTAINING CARDIAC OUTPUT

Cardiac output is maintained (determined) by four factors:

1. Venous return
2. Force of contraction
3. Heart rate
4. Peripheral resistance.

■ 1. VENOUS RETURN

Venous return is the amount of blood which is returned to heart from different parts of the body. When it increases, the ventricular filling and cardiac output are increased. Thus, cardiac output is **directly proportional** to venous return, provided the other factors (force of contraction, heart rate and peripheral resistance) remain constant.

Venous return in turn, depends upon five factors:

i. Respiratory pump
ii. Muscle pump
iii. Gravity
iv. Venous pressure
v. Sympathetic tone.

i. *Respiratory Pump*

Respiratory pump is the respiratory activity that helps the return of blood, to heart during inspiration. It is also called **abdominothoracic pump.** During inspiration, thoracic cavity expands and makes the intrathoracic pressure more negative. It increases the diameter of inferior vena cava, resulting in increased venous return. At the same time, descent of diaphragm increases the intra-abdominal pressure, which compresses abdominal veins and pushes the blood upward towards the heart and thereby the venous return is increased (Fig. 98.1).

Respiratory pump is much stronger in forced respiration and in severe muscular exercise.

TABLE 98.1: Distribution of blood pumped out of left ventricle

Organ	Amount of blood (mL/ minute)	Percentage
Liver	1,500	30
Kidney	1,300	26
Skeletal muscles	900	18
Brain	800	16
Skin, bone and GI tract	300	6
Heart	200	4
Total	5,000	100

ii. *Muscle Pump*

Muscle pump is the muscular activity that helps in return of the blood to heart. During muscular activities, the veins are compressed or squeezed. Due to the presence of valves in veins, during compression the blood is moved towards the heart (Fig. 98.2). When muscular activity increases, the venous return is more.

When the skeletal muscles contract, the vein located in between the muscles is compressed. Valve of the vein

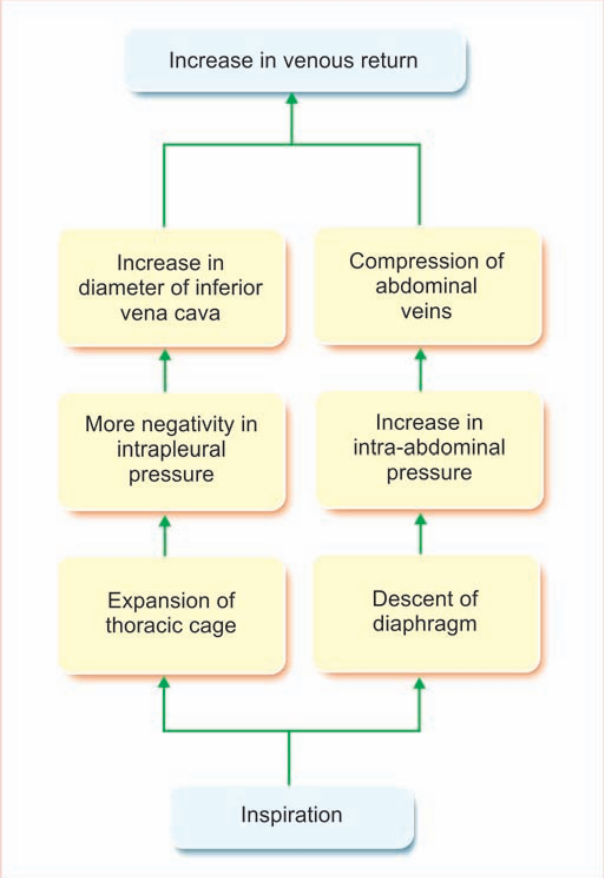

Increase in venous return
Increase in diameter of inferior vena cava
Compression of abdominal veins
More negativity in intrapleural pressure
Increase in intra-abdominal pressure
Expansion of thoracic cage
Descent of diaphragm
Inspiration

FIGURE 98.1: Effect of respiratory pump on venous return

proximal to the contracting muscles (Fig. 98.2 A) is opened and the blood is propelled towards the heart. Valve of the vein distal to the muscles is closed by the back flow of blood.

During relaxation of the muscles (Fig. 98.2 B), the valve proximal to muscles closes and prevents the back flow of blood. The valve distal to the muscles opens and allows the blood to flow upwards.

iii. *Gravity*

Gravitational force reduces the venous return. When a person stands for a long period, gravity causes pooling of blood in the legs, which is called **venous pooling.** Because of venous pooling, the amount of blood returning to heart decreases.

iv. *Venous Pressure*

Venous pressure also affects the venous return. Pressure in the venules is 12 to 18 mm Hg. In the smaller and larger veins, the pressure gradually decreases. In the great veins, i.e. inferior vena cava and superior vena cava, the pressure falls to about 5.5 mm Hg. At the junction of venae cavae and right atrium, it is about 4.6 mm Hg. Pressure in the right atrium is still low and it alters during cardiac action. It falls to zero during atrial diastole. This pressure gradient at every part of venous tree helps as a driving force for venous return.

v. *Sympathetic Tone*

Venous return is aided by sympathetic or vasomotor tone (Chapter 103), which causes constriction of venules. Venoconstriction pushes the blood towards heart.

■ 2. FORCE OF CONTRACTION

Cardiac output is **directly proportional** to the force of contraction, provided the other three factors remain constant. According to **Frank-Starling law,** force of contraction of heart is directly proportional to the initial length of muscle fibers, before the onset of contraction.

Force of contraction depends upon preload and afterload.

Preload

Preload is the stretching of the cardiac muscle fibers at the end of diastole, just before contraction. It is due to increase in ventricular pressure caused by filling of blood during diastole. Stretching of muscle fibers increases their length, which increases the force of contraction and cardiac output.

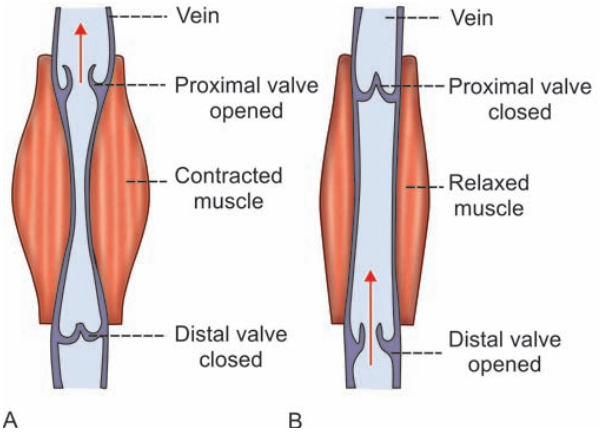


FIGURE 98.2: Mechanism of muscle pump. A. During contraction of the muscle; B. During relaxation of the muscle.

Thus, force of contraction of heart and cardiac output are **directly proportional** to preload.

Afterload

Afterload is the force against which ventricles must contract and eject the blood. Force is determined by the arterial pressure. At the end of isometric contraction period, semilunar valves are opened and blood is ejected into the aorta and pulmonary artery. So, the pressure increases in these two vessels. Now, the ventricles have to work against this pressure for further ejection. Thus, the afterload for left ventricle is determined by aortic pressure and afterload for right ventricular pressure is determined by pressure in pulmonary artery.

Force of contraction of heart and cardiac output are **inversely proportional** to afterload.

■ 3. HEART RATE

Cardiac output is **directly proportional** to heart rate provided, the other three factors remain constant. Moderate change in heart rate does not alter the cardiac output. If there is a marked increase in heart rate, cardiac output is increased.

If there is marked decrease in heart rate, cardiac output is decreased.

■ 4. PERIPHERAL RESISTANCE

Peripheral resistance is the resistance offered to blood flow at the peripheral blood vessels. Peripheral resistance is the resistance or load against which the heart has to pump the blood. So, the cardiac output is **inversely proportional** to peripheral resistance.

Resistance is offered at **arterioles** so, the arterioles are called **resistant vessels.** In the body, maximum peripheral resistance is offered at the **splanchnic region.** Other details of peripheral resistance are given in Chapter 102.

MEASUREMENT OF CARDIAC OUTPUT

Cardiac output is measured by direct methods and indirect methods. Direct methods are used only in animals. Indirect methods are used both in animals and human beings.

MEASUREMENT OF CARDIAC OUTPUT BY DIRECT METHODS

Direct methods used to measure cardiac output in animals:

1. By using cardiometer
2. By using flowmeter.

1. *By Using Cardiometer*

This is described in Chapter 91.

2. *By Using Flowmeter*

Mechanical flowmeter

Mechanical flowmeter is used to measure cardiac output or the amount of blood flow to any organ. It is used only in animals. It has an inlet, a measuring device in the middle and an outlet. Aorta or the artery entering any organ is cut. Inlet and outlet of the flowmeter are inserted into cut ends of the blood vessel. When the blood passes through the flowmeter, the measuring device determines the amount of blood flow (Fig. 99.1).

Electromagnetic flowmeter

Principle: Principle of this flowmeter is to develop an electromagnetic field by means of two coils of wire. If the coils are placed on either side of a blood vessel, the electromagnetic field is produced around the vessel. When blood flows through the vessel, there is an alteration in the electromagnetic field. By using appropriate electrodes, the changes in the magnetic field can be detected. By connecting electrodes to an electronic device, velocity of blood flow is determined on the basis of changes in the magnetic field. From the velocity of blood flow, the volume of blood flow is calculated.

Instrument: An **electromagnetic probe** is devised with the electromagnetic coils and the electrodes. The probe has a cleft and it is fixed in such a way that the intact blood vessel passes through the cleft. The probe almost encircles the blood vessel. The probe is connected to the electronic device to measure the volume of blood flow.

Advantage of this flowmeter is that the blood vessel need not be cut open.

Ultrasonic Doppler flowmeter

Principle: Ultrasound is the sound with very high frequency. It is very much beyond the audible range of human ears. The waves of the **ultrasound** are transmitted through a blood vessel. These sound waves are called **transmitted waves.** While passing through the blood vessels, the sound waves hit against the blood cells, particularly the red blood cells and are reflected back. Frequency of the **reflected waves** is different from that of the transmitted waves. This effect is called the **Doppler effect** (named after the discoverer **Johann Christian Doppler**). Alteration in the frequency of reflected waves depends upon the velocity of blood flowing through the blood vessel. By detecting the differences between frequencies of transmitted and reflected sound waves, the velocity of blood flow and then the volume of blood flow are determined.

Instrument: Ultrasonic device has piezoelectric crystals, which produce the **ultrasonic waves** and act as sensors to receive the reflected waves. This device is connected to an electronic equipment, which detects the difference between the frequencies of transmitted and reflected waves and thereby, determines the velocity of blood flow and the volume of blood flow.

Disadvantages of Direct Methods

i. Direct methods to measure cardiac output can be used only in animals
ii. Blood vessel has to be cut open at the risk of animal's life
iii. While using cardiometer, the size of the cardiometer must be suitable for the size of the heart
iv. While using mechanical flowmeter, diameter of inlet and the outlet of the flowmeter must be equivalent to the diameter of the blood vessel.

MEASUREMENT OF CARDIAC OUTPUT BY INDIRECT METHODS

Several methods are available to measure cardiac output. Each method has got its own advantages and disadvantages. Generally, the safe and accurate method is preferred. In view of safety, always non-invasive methods are preferred. The invasive method

is also accepted provided, it gives accurate results. In addition to providing measurement of cardiac output, nowadays the methods are expected to provide other hemodynamic data and some useful information about the structure and movements of valves and chambers of the heart.

Invasive and Non-invasive Methods

Invasive method refers to a procedure which involves invasion or penetration of healthy tissues, organs or parts of the body, by means of perforation, puncture, incision, injection or catheterization. Non-invasive method means the procedure that does not involve invasion or penetration of tissues, organs or parts of the body.

Different Indirect Methods

Indirect methods used to measure cardiac output:

1. By using Fick principle
2. Indicator (dye) dilution technique
3. Thermodilution technique
4. Ultrasonic Doppler transducer technique
5. Doppler echocardiography
6. Ballistocardiography.

1. *By Using Fick Principle*

Adolph Fick described Fick principle in 1870. According to this principle, the amount of a substance taken up by an organ (or by the whole body) or given out in a unit of time is the product of amount of blood flowing through the organ and the arteriovenous difference of the substance across the organ.

Amount of substance taken or given = Amount of blood flow/minute × Arteriovenous difference

For example,

Amount of blood flowing through lungs is 5,000 mL/minute

O_2 content in arterial blood = 20 mL/100 mL of blood

O_2 content in venous blood = 15 mL/100 mL of blood

Amount of oxygen moved from lungs to blood = Amount of blood flow/minute × Arteriovenous difference of O_2

$$= 5{,}000 \times \frac{20 - 15}{100}$$

$$= 5{,}000 \times \frac{5}{100} = 250$$

Amount of oxygen moved from lungs to blood = 250 mL/minute

Modification of Fick principle to measure cardiac output

Fick principle is modified to measure the cardiac output or a part of cardiac output (amount of blood to an organ). Thus, cardiac output or the amount of blood flowing through an organ in a given unit of time is determined by the formula:

$$\text{Cardiac output} = \frac{\text{Amount of substance taken or given by the organ/minute}}{\text{Arteriovenous difference of the substance across the organ}}$$

By modifying Fick principle, cardiac output is measured in two ways:

i. By using oxygen consumption
ii. By using carbon dioxide given out.

Measurement of Cardiac Output by Using Oxygen Consumption

Fick principle is used to measure the cardiac output by determining the amount of oxygen consumed in the body in a given period of time and dividing this value by the arteriovenous difference across the lungs.

$$\text{Cardiac output} = \frac{O_2 \text{ consumed (in mL/minute)}}{\text{Arteriovenous } O_2 \text{ difference}}$$

Oxygen consumption: Amount of oxygen consumed is measured by using a **respirometer** or **BMR apparatus (Benedict Roth apparatus).**

Oxygen content in arterial blood: Blood is collected from any artery to determine the oxygen content in arterial blood. Oxygen content is determined by blood gas analysis.

Oxygen content in venous blood: Only mixed venous blood is used to determine the oxygen content of venous blood, since oxygen content is different in different veins. Mixed venous blood is collected from right atrium or pulmonary artery. It is done by introducing a **catheter** through basilar vein of forearm. Oxygen is determined from this blood by **blood gas analysis** (Fig. 98.3).

Calculation

For example, in a subject, the following data are obtained:

O_2 consumed (by lungs) = 250 mL/minute
O_2 content in arterial blood = 20 mL/100 mL of blood
O_2 content in venous blood = 15 mL/100 mL of blood

$$\text{Cardiac output} = \frac{O_2 \text{ consumed (in mL/minute)}}{\text{Arteriovenous } O_2 \text{ difference}}$$

$$= \frac{250}{5/100} = \frac{250 \times 100}{5}$$

$$= 5{,}000 \text{ mL/minute}$$

5 mL of oxygen is taken by 100 mL of blood while passing through the lungs. Thus, 250 mL of oxygen is taken by 5,000 mL of blood. Since cardiac output is equivalent to the amount of blood passing through pulmonary circulation, the cardiac output = 5 L/minute.

Measurement of Cardiac Output by Using Carbon Dioxide

Cardiac output is also measured by knowing the arteriovenous difference of carbon dioxide and amount of carbon dioxide given out (removed) by lungs (Fig. 98.4). Thus:

$$\text{Cardiac output} = \frac{CO_2 \text{ evolved (in mL/minute)}}{\text{Arteriovenous } CO_2 \text{ difference}}$$

Calculation

For example, in a subject

CO_2 removed by lungs = 200 mL/minute

CO_2 content in arterial blood = 56 mL/100 mL of blood

CO_2 content in venous blood = 60 mL/100 mL of blood

$$\text{Cardiac output} = \frac{200}{60 - 56 \text{ mL}/100 \text{ mL}}$$

$$= \frac{200 \times 100}{4}$$

$$= 5{,}000 \text{ mL} = 5 \text{ L/minute}$$

Since cardiac output is equal to the amount of blood passing through lungs (pulmonary circulation), the cardiac output = 5 L/minute

Nitrous oxide is also used to measure cardiac output by applying Fick principle.

Advantage of measurement of cardiac output by Fick principle

The results are accurate.

Disadvantage

Fick principle is an invasive method and involves the insertion of catheter through subject vein.

2. Indicator (Dye) Dilution Method

Indicator dilution technique is described in detail in Chapter 6. Marker substance used to measure cardiac output is lithium chloride.

Advantage

The results are accurate.

Disadvantage

Indicator dilution method is an invasive method and involves injection of marker substance.

3. Thermodilution Technique

Cardiac output can also be measured by thermodilution technique or **thermal indicator method.** This method is the modified indicator dilution method. It is the popular method to measure cardiac output.

In this method, a known volume of cold sterile solution is injected into the right atrium via inferior vena

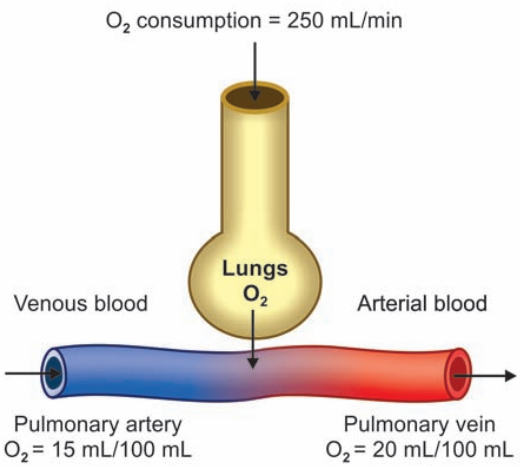


FIGURE 98.3: Oxygen consumption

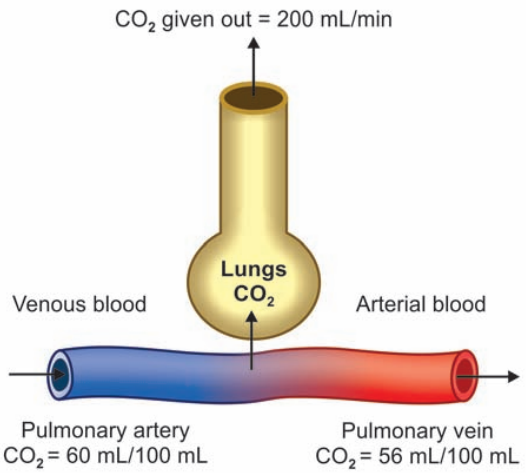


FIGURE 98.4: Carbon dioxide given out

cava by using a catheter. Cardiac output is measured by determining the resultant change in the blood temperature in pulmonary artery. For this purpose, two **thermistors (temperature transducers)** are used. One of them is placed in the inferior vena cava and the second one is placed in pulmonary artery. A pulmonary artery catheter is used to place the thermistors in their positions.

A known quantity of cold saline or cold dextrose solution is injected into inferior vena cava. Thermistors determine the temperature of blood entering the heart via inferior vena cava and temperature of blood leaving the heart via pulmonary artery. From the values of temperature, cardiac output is measured by applying indicator dilution technique.

Advantages

Results are accurate in this method. Even low cardiac output can be measured. Saline is also harmless. Catheter is also used to determine hemodynamic pressures and to collect mixed venous blood.

Disadvantage

Thermodilution technique is an invasive method and it requires catheterization.

Continuous cardiac output measurement catheter

Cardiac output can be measured continuously by using a modified pulmonary artery catheter called **continuous cardiac output measurement catheter** (CCO catheter). CCO catheter works on thermodilution principle. Instead of injecting cold saline, a heating filament which delivers heat directly to blood is used. The heating filament is fitted to the ventricular portion of the catheter. Cardiac output is measured as done in thermodilution technique. This method is commonly used in intensive care unit (ICU).

4. *Esophageal Ultrasonic Doppler Transducer Technique*

Esophageal ultrasonic doppler transducer technique involves insertion of a flexible probe into midthoracic part of esophagus. A pulse wave ultrasonic **Doppler transducer** is fixed at the tip of the probe. This transducer calculates the velocity of blood flow in descending aorta (refer ultrasonic Doppler flow meter for details). The diameter of aorta is determined by echocardiography (see below). Cardiac output is calculated by using the values of velocity of blood flow and diameter of aorta.

Advantages

The cardiac output can be measured continuously. This can be used during cardiac surgery.

Disadvantages

Esophageal ultrasonic doppler transducer is an invasive method and results are less accurate.

5. *Doppler Echocardiography*

Doppler echocardiography is a method for detecting the direction and velocity of moving blood within the heart. This is also a popular method to measure cardiac output.

Echocardiography is a **diagnostic procedure,** which uses the ultrasound waves (more than 20,000 Hz) to produce the image of the heart muscle. Ultrasound waves which reflect or echo off the heart can determine the size, shape, movement of the valves and chambers and the flow of blood through the heart.

During echocardiographic examination, the patient lies bare-chested on the examination table. A special gel is spread over the chest to help the transducer make good contact and slide smoothly over the skin. The transducer is a small hand operated device, which is attached to machine by a flexible cable. The transducer is placed against the chest. The transducer produces and directs ultrasound waves into the chest. Some of the waves get reflected (or echoed) back to the transducer. The reflection of sound waves depends upon the type of tissues and blood. The reflected sound waves are received by the transducer and translated into an image of the heart and displayed on a monitor or recorded on paper or tape.

Echocardiography may also show the abnormalities in functioning of heart valves or damage to the myocardium from an earlier heart attack.

When **Doppler principle** is applied in echocardiography, it enables the determination of direction, rate and other characteristics of blood flow. Doppler echocardiography is based upon the changes in frequency of the reflected sound waves from red blood cells (refer ultrasonic Doppler flow meter for details).

By Doppler echocardiography, the velocity of blood flow through aortic valve is determined. The diameter of the aorta is determined by simple echocardiography. From these values, cardiac output is calculated.

Advantage

Doppler echocardiography is a non-invasive technique. It also provides other useful information about the structures and movements of valves and chambers of heart.

Disadvantage

Doppler echocardiography method provides less accurate results. It requires well trained operator.

6. *Ballistocardiographic Method*

Ballistocardiography is the technique to record the movements of the body caused by **ballistic recoil,** associated with contraction of heart and ejection of blood. It is based on **Newton's third law of motion** (for every action there is an equal and opposite reaction). When heart pumps blood into aorta and pulmonary artery, a recoiling force is exerted against heart and the body. It is similar to that of ballistic recoil when a bullet is fired from a riffle.

Pulsations due to this ballistic recoil can be recorded graphically by making the subject to lie on a suspended bed, movable in the long axis of the body. The cardiac output is determined by analyzing the graph obtained.

Advantage

The only advantage of ballistocardiography is that it is a non-invasive method.

Disadvantage

Ballistocardiography is not a commonly used technique because it involves cumbersome procedures for calibrating the equipment and analyzing the graph. It also does not provide accurate results.

■ CARDIAC CATHETERIZATION

■ DEFINITION

Catheter is a thin radiopaque tube, made up of elastic web, rubber, plastic, glass or metal. Cardiac catheterization is an invasive procedure in which a catheter is inserted **intravascularly** into any chamber of the heart or a blood vessel.

Cardiac catheterization is helpful to study the different variables of hemodynamics, both in normal and diseased states. Cardiac catheterization was discovered by a German medical student Werner Forsmann, who practiced this technique first on himself.

■ CONDITIONS WHEN CARDIAC CATHETERIZATION IS PERFORMED

Cardiac catheterization is generally performed:

1. When clinical assessments indicate rapid deterioration of patient's health and immediate treatment. This is the most common condition when cardiac catheterization is needed.
2. Whenever there is a need to confirm the suspected cardiac disease of a patient
3. Whenever there is need to determine anatomical and physiological status of heart and blood vessels.

■ PROCEDURE

Cardiac catheterization is performed by insertion of catheter into the peripheral blood vessel through skin, by needle puncture. This procedure is called **percutaneous insertion** of catheter.

Left Heart Catheterization

Left heart catheterization is done by passing a catheter through femoral artery, brachial artery or axillary artery. Catheter is guided into left ventricle under fluoroscopic observation via aorta. From left ventricle, the catheter is advanced into left atrium.

In patients with aortic stenosis or **prosthetic** (artificial) **valve,** the direct left ventricular puncture is performed. Under local anesthesia, a needle with a catheter is inserted through the thoracic wall at the level of apex beat. When the needle enters left ventricle, the catheter is advanced through the needle into left ventricle and later the needle is removed.

Latest technology includes catheterization through radial artery, which is called **transradial catheterization.**

Right Heart Catheterization

Right heart catheterization is usually performed by venous puncture via femoral vein. Catheter can also be introduced via internal jugular vein, subclavian vein or medial vein. Under **fluoroscopic observation,** the catheter is advanced into right atrium. From right atrium, it can be guided into right ventricle and also into pulmonary artery.

■ USES OF CARDIAC CATHETERIZATION

Cardiac catheterization is useful for both diagnostic and therapeutic purposes. It gives crucial information about the need for cardiac surgery, coronary angioplasty and other **therapeutic procedures.** It also gives information about anticipated risks and reversibility in the patient's condition during **cardiac surgery** or other **therapeutic interventions.**

Diagnostic Uses of Cardiac Catheterization

1. Blood samples are collected during cardiac catheterization to measure oxygen saturation and the concentration of ischemic metabolites like lactate
2. Cardiac output is measured by using Fick principle, indicator dilution technique or thermodilution technique during cardiac catheterization

3. Angiography is done with the help of catheterization. Angiography or **arteriography** is the diagnostic or **therapeutic radiography (imaging technique),** in which the fluoroscopic picture is used to visualize the blood filled structures like cardiac chambers, arteries and veins of heart and other blood vessels, by using a **radiopaque contrast** medium. It is used to determine the obstruction or occlusion of coronary blood vessels or other blood vessels. It is also used to determine the anomalies of coronary blood vessels.
4. Various pressures are determined by attaching a pressure transducer to the cardiac catheter.

   Right heart catheterization is used to measure:
   i. Right atrial pressure
   ii. Right ventricular pressure
   iii. Pulmonary arterial pressure
   iv. Pulmonary capillary wedge pressure.

   Left heart catheterization is used to measure:
   i. Aortic pressure
   ii. Left ventricular pressure
   iii. Left atrial pressure.

Therapeutic Uses of Cardiac Catheterization – Interventional Cardiology

Cardiac catheterization is performed for various therapeutic procedures. Interventional cardiology is a branch of cardiology that deals with performance of traditional surgical procedures by cardiac catheterization. It helps in:

1. Thrombolysis
2. Percutaneous transluminal coronary angioplasty
3. Laser coronary angioplasty
4. Catheter ablation.

1. *Thrombolysis*

Thrombolysis **(reperfusion therapy)** is the procedure used to break up and dissolve a **thrombus** (clot) in the coronary artery of patient affected by acute myocardial infarction due to coronary thrombus. Cardiac catheterization is used for intracoronary administration of **thrombolytic agents** which cause thrombolysis.

Thrombolytic agents:
i. Tissue plasminogen activator
ii. Streptokinase
iii. Urokinase.

All these thrombolytic agents convert plaminogen into plasmin, which degrades fibrin in clot and restore normal blood flow.

2. *Percutaneous transluminal coronary angioplasty*

Coronary angioplasty means the correction of narrowed or totally obstructed lumen of blood vessels by mechanical methods. In percutaneous transluminal coronary angioplasty (PTCA), a narrowed coronary artery is dilated by inflating a balloon attached to the tip of catheter that is introduced into the blood vessel. Sometimes, a **stent** (expandable wire mesh) is introduced into the corrected blood vessel by the catheter to keep the vessel in dilated state.

3. *Laser coronary angioplasty*

Catheter is also used to emit laser (Light amplification by stimulated emission of radiation) energy. Laser energy which is emitted into the occluded coronary artery vaporizes the atherosclerotic plaque in the diseased vessel. This technique is called laser coronary angioplasty.

4. *Catheter ablation*

Catheter ablation is the procedure to destroy (ablate) an area of cardiac tissue that blocks the electrical pathway or produces abnormal electrical impulses, resulting in cardiac arrhythmia such as supraventricular tachycardia (SVT) or Wolff-Parkinson-White syndrome (Chapter 96).

It involves advancing a catheter (with electrodes attached to its tip) towards the heart via either femoral vein or subclavian vein. When the catheter enters right atrium, arrhythmia is induced. Then the electrodes at the tip of catheter record the electrical potentials. By using these recordings, the area of faulty electrical site is pinpointed. This procedure is called **electrical mapping.**

Once the damaged site is confirmed, **radiofrequency energy** is used to destroy the small amount of tissue that disturbs the electrical flow through the heart. Thus, the healthy heart rhythm is restored. Tissue is also destroyed by freezing with intense cold **(cryoablation).**

Heart-lung Preparation

Chapter 99

- INTRODUCTION
- PROCEDURE
- USES OF HEART-LUNG PREPARATION

INTRODUCTION

Heart-lung preparation is an experimental set up, devised by **Starling.** It is used to demonstrate the effects of various factors on the activities of heart, particularly heart rate and cardiac output. This preparation is also used to record the cardiac function curves.

PROCEDURE

Heart-lung preparation is usually done in dogs. After giving **anesthesia,** neck of the dog is opened and a **tracheal cannula** is inserted into the trachea. Tracheal cannula is connected to a **respiratory pump,** so that respiration in the animal is controlled artificially, to avoid any disturbance during the experimental procedure (Fig. 99.1).

Then, chest is opened and an **arterial cannula** is inserted into one of the branches of aorta. All the other branches from arch of aorta and descending aorta are ligated. Arterial cannula is connected to two instruments:

1. **Mercury manometer** to measure the arterial blood pressure
2. **Air bottle,** which provides elasticity artificially (as in the case of arterial wall).

Thus, the blood ejected from left ventricle passes into air bottle through the arterial cannula and rubber tubes. From the air bottle, the blood is diverted through a tube which provides **artificial resistance.** Air bottle is also connected to a **pressure bottle.** Pressure bottle is attached to a pressure pump. This pump is used to maintain the pressure within the set up.

Artificial resistance is offered by applying pressure surrounding the **resistance tube.** Resistance tube is also connected to a manometer.

After passing through the resistance tube, blood is allowed to flow through a **warming glass coil,** which is kept inside a water bath with a heater. Temperature of water bath is controlled, so that the temperature of blood could be maintained.

Warming coil is connected to a **venous reservoir** through a flowmeter, which determines the amount of blood flow (cardiac output). Venous reservoir is connected to superior vena cava by a rubber tube. A screw type clamp is fitted to the rubber tube. This clamp is used to adjust the amount of blood returning to heart (venous return). A **thermometer** is also fitted to the tube to note the temperature of blood.

A third mercury manometer is connected to the inferior vena cava. It is used to determine the venous pressure. A cardiometer is fitted to the ventricle. This cardiometer is connected to a recording device like **Marey tambour** or **polygraph,** to record the ventricular volume changes.

Pulmonary circulation is kept intact for continuous oxygenation of blood.

USES OF HEART-LUNG PREPARATION

Thus, in this set up, the heart works as an isolated organ. So, the effects of various factors can be demonstrated on the activities of heart, like heart rate, ventricular volume and cardiac output.

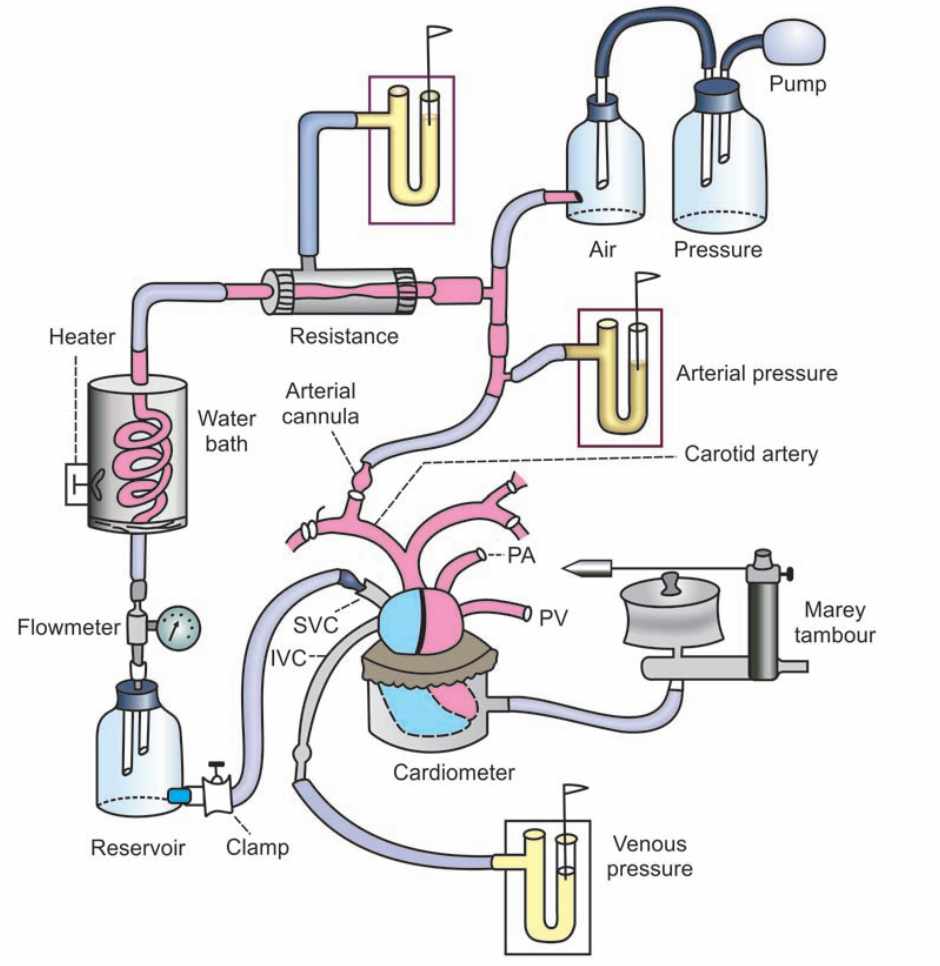


FIGURE 99.1: Heart-lung preparation.
SVC = Superior vena cava, IVC = Inferior vena cava, PA = Pulmonary artery, PV = Pulmonary vein.

Examples

1. When venous return decreases, stroke volume decreases
2. When venous return increases, stroke volume increases
3. When resistance increases, cardiac output decreases
4. When resistance decreases, cardiac output increases
5. Heart-lung preparation is also used to record two types of cardiac function curves:
   i. Cardiac output curves
   ii. Venous return curves.

Though the cardiac function curves are obtained in experiments using the animals, these curves represent the functions of the ventricles in human heart also (Chapter 100).

Chapter 100

Cardiac Function Curves

- INTRODUCTION
- CARDIAC OUTPUT CURVES
  - NORMAL CARDIAC OUTPUT CURVES
  - FACTORS AFFECTING CARDIAC OUTPUT CURVES
  - EFFECT OF EXTRACARDIAC PRESSURE ON CARDIAC OUTPUT CURVE
- VENOUS RETURN CURVES
- ANALYSIS OF CARDIAC FUNCTION CURVES
  - COUPLING OF CARDIAC AND VASCULAR FUNCTIONS

INTRODUCTION

Cardiac function curves are **Frank-Starling curves,** which demonstrate the capacity of ventricles to pump blood and to maintain blood circulation throughout the body. Most of the cardiac function curves are obtained from animal experiments, by using heart-lung preparation. However, these curves are considered to represent the functions of ventricles in human heart.

Cardiac function curves are of two types:

1. Cardiac output curves
2. Venous return curves.

CARDIAC OUTPUT CURVES

Cardiac output curves are the curves that show the relationship between cardiac output and right atrial pressure. Right atrial pressure, in turn, depends upon venous return.

NORMAL CARDIAC OUTPUT CURVES

Normally, left ventricular output is 5 L/minute, when the pressure in right atrium is 2 mm Hg. When the atrial pressure rises between 4 and 8 mm Hg, the left ventricular output also increases. It increases to about two and a half times of normal (basal) output, i.e. the output increases to about 13 to 14 L/minute. This is the maximum limit for increase in cardiac output. Further increase in right atrial pressure does not increase the ventricular output and the curve shows a **plateau** (Fig. 100.1).

Right ventricular output is 5 L/minute, when the right atrial pressure is zero. This reaches the maximum, i.e. 13 to 14 L/minute when the atrial pressure increases between 2 and 4 mm Hg (Fig. 100.1).

Thus, the cardiac output curves demonstrate that cardiac output is directly proportional to atrial pressure up to a certain extent (as explained above).

Plateau of the curve shows that the heart can control the output by itself if the atrial pressure rises beyond +8 mm Hg. It is due to the fact that in normal conditions, venous return is decreased when atrial pressure raises above +8 mm Hg.

FACTORS AFFECTING CARDIAC OUTPUT CURVES

Shifting of cardiac output curve to left indicates increase in cardiac output and shifting to right indicates decrease in cardiac output. The conditions which shift the cardiac output curve to left or right are discussed below:

Shift to Left

When there is an abnormal increase in the functioning of the heart **(hypereffective heart),** the cardiac output curve is shifted to left, indicating **increase in cardiac output.**

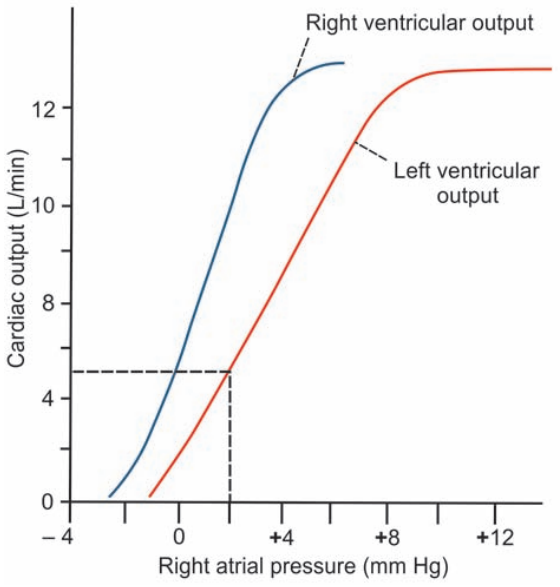


FIGURE 100.1: Normal cardiac output curves

Conditions when shift to left occurs

1. Combined stimulation of sympathetic and the parasympathetic nerves supplying the heart: It causes hyperexcitation of the heart, resulting in increased rate and force of contraction. The cardiac output increases up to 25 L/minute (i.e. the plateau is shifted to left). Increase in output is about twice the maximum output in normal conditions (13 to 14 L/minute).
2. Hypertrophy of heart: It increases cardiac output up to 10 to 19 L/minute. It is because of increase in force of contraction.
3. Excitation (by cardiac nerves) of the heart along with hypertrophy of the ventricles: In this condition, the cardiac output is elevated above 35 L/minute. It occurs in Marathon runners. Increase in cardiac output is an important factor for prolonged running time of Marathon runners.

Shift to Right

When the functioning of heart decreases **(hypoeffective heart),** cardiac output curve is shifted to right, indicating **decrease in cardiac output.**

Conditions when shift to right occurs

1. Stimulation of parasympathetic nerve fibers of the heart
2. Inhibition of sympathetic nerves to heart
3. Myocardial infarction
4. Diseases of the valves in the heart
5. Congenital heart diseases.

■ EFFECT OF EXTRACARDIAC PRESSURE ON CARDIAC OUTPUT CURVE

Extracardiac pressure is the pressure outside the heart. Intrapleural pressure is the major extracardiac pressure. When it increases above the normal level, i.e. from –6 to –2 mm Hg or becomes positive, the venous return decreases, resulting in decrease in cardiac output. Cardiac output curve is shifted to right. It happens in opening of thoracic cage and in positive pressure breathing.

When the intrapleural pressure decreases, i.e. when it becomes more negative, the venous return increases and the cardiac output also increases. The curve is shifted towards left. It is common in **negative pressure breathing.**

Cardiac Tamponade

Cardiac tamponade is the mechanical compression of heart due to accumulation of fluid in **pericardial space.** In addition to intrapleural pressure, accumulation of fluid in pericardial space also increases the extracardiac pressure and compresses the heart. In cardiac tamponade, the cardiac output decreases and output curve is shifted to right.

■ VENOUS RETURN CURVES

Venous return curves are the curves which demonstrate the relationship between venous return (blood flow in vascular system) and right atrial pressure. Venous return curves are also called **systemic vascular function curves.**

Normally, 5 L of blood returns to heart every minute. When right atrial pressure increases, venous return decreases due to **backpressure.** When venous return decreases, the cardiac output also decreases (Fig. 100.2).

■ ANALYSIS OF CARDIAC FUNCTION CURVES

Relation of cardiac output and venous return with right atrial pressure is determined when cardiac output curves and venous return curves are merged together (Fig. 100.3).

■ COUPLING OF CARDIAC AND VASCULAR FUNCTIONS

Cardiac output represents cardiac function and venous return represents vascular function. Coupling or merging of cardiac output (cardiac function) curves and venous return (vascular function) curves shows that when venous return is normal (5 L/minute), the cardiac output as well as the right atrial pressure are normal.

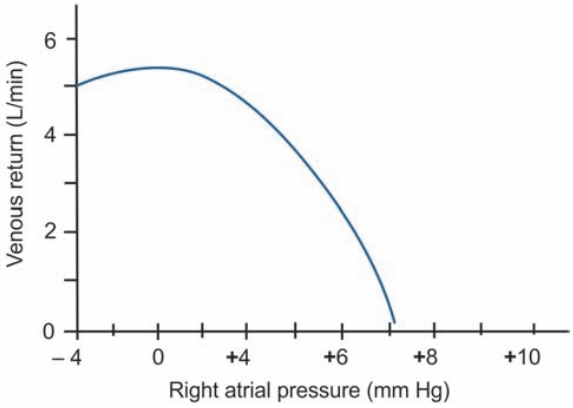


FIGURE 100.2: Venous return curve

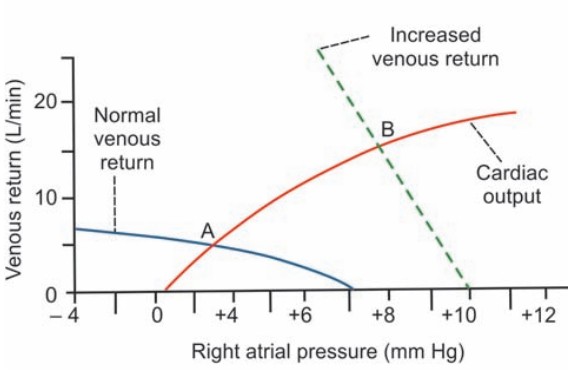


FIGURE 100.3: Analysis of cardiac function curves

Relation between cardiac output and venous return under normal conditions is represented by (A). When the venous return increases (B), the cardiac output also increases along with increase in right atrial pressure. Thus, any factor that alters venous return alters the cardiac output also.

Heart Rate

Chapter 101

- HEART RATE
  - NORMAL HEART RATE
  - TACHYCARDIA
  - BRADYCARDIA
- REGULATION OF HEART RATE
- VASOMOTOR CENTER – CARDIAC CENTER
  - VASOCONSTRICTOR AREA
  - VASODILATOR AREA
  - SENSORY AREA
- MOTOR (EFFERENT) NERVE FIBERS TO HEART
  - PARASYMPATHETIC NERVE FIBERS
  - SYMPATHETIC NERVE FIBERS
- SENSORY (AFFERENT) NERVE FIBERS FROM HEART
- FACTORS AFFECTING VASOMOTOR CENTER – REGULATION OF VAGAL TONE
  - IMPULSES FROM HIGHER CENTERS
  - IMPULSES FROM RESPIRATORY CENTERS
  - IMPULSES FROM BARORECEPTORS
  - IMPULSES FROM CHEMORECEPTORS
  - IMPULSES FROM RIGHT ATRIUM
  - IMPULSES FROM OTHER AFFERENT NERVES
  - BEZOLD-JARISCH REFLEX

HEART RATE

NORMAL HEART RATE

Normal heart rate is 72/minute. It ranges between 60 and 80 per minute.

TACHYCARDIA

Tachycardia is the increase in heart rate above 100/minute.

Physiological Conditions when Tachycardia Occurs

1. Childhood
2. Exercise
3. Pregnancy
4. Emotional conditions such as anxiety.

Pathological Conditions when Tachycardia Occurs

1. Fever
2. Anemia
3. Hypoxia
4. Hyperthyroidism
5. Hypersecretion of catecholamines
6. Cardiomyopathy
7. Diseases of heart valves.

■ BRADYCARDIA

Bradycardia is the decrease in heart rate below 60/minute.

Physiological Conditions when Bradycardia Occurs

1. Sleep
2. Athletes.

Pathological Conditions when Bradycardia Occurs

1. Hypothermia
2. Hypothyroidism
3. Heart attack
4. Congenital heart disease
5. Degenerative process of aging
6. Obstructive jaundice
7. Increased intracranial pressure.

Drugs which Induce Bradycardia

1. Beta blockers
2. Channel blockers
3. Digitalis and other antiarrhythmic drugs.

■ REGULATION OF HEART RATE

Heart rate is maintained within normal range constantly. It is subjected for variation during normal physiological conditions such as exercise, emotion, etc. However, under physiological conditions, the altered heart rate is quickly brought back to normal. It is because of the perfectly tuned regulatory mechanism in the body.

Heart rate is regulated by the nervous mechanism, which consists of three components:

A. Vasomotor center
B. Motor (efferent) nerve fibers to the heart
C. Sensory (afferent) nerve fibers from the heart.

■ VASOMOTOR CENTER – CARDIAC CENTER

Vasomotor center is the nervous center that regulates the heart rate. It is the same center in brain, which regulates the blood pressure. It is also called the cardiac center.

Vasomotor center is bilaterally situated in the **reticular formation** of medulla oblongata and lower part of pons.

Areas of Vasomotor Center

Vasomotor center is formed by three areas:

1. Vasoconstrictor area
2. Vasodilator area
3. Sensory area.

■ VASOCONSTRICTOR AREA – CARDIOACCELERATOR CENTER

Situation

Vasoconstrictor area is situated in the reticular formation of medulla in floor of IV ventricle and it forms the lateral portion of vasomotor center. It is otherwise known as **pressor area** or cardioaccelerator center.

Function

Vasoconstrictor area increases the heart rate by sending accelerator impulses to heart, through **sympathetic nerves.** It also causes constriction of blood vessels. Stimulation of this center in animals increases the heart rate and its removal or destruction decreases the heart rate.

Control

Vasoconstrictor area is under the control of **hypothalamus** and **cerebral cortex.**

■ VASODILATOR AREA – CARDIOINHIBITORY CENTER

Situation

Vasodilator area is also situated in the reticular formation of medulla oblongata in the floor of IV ventricle. It forms the medial portion of vasomotor center. It is also called **depressor area** or cardioinhibitory center.

Function

Vasodilator area decreases the heart rate by sending inhibitory impulses to heart through vagus nerve. It also causes dilatation of blood vessels. Stimulation of this area in animals with weak electric stimulus decreases the heart rate and stimulation with a strong stimulus stops the heartbeat. When this area is removed or destroyed, heart rate increases.

Control

Vasodilator area is under the control of **cerebral cortex** and **hypothalamus.** It is also controlled by the impulses from baroreceptors, chemoreceptors and other sensory impulses via afferent nerves.

SENSORY AREA

Situation

Sensory area is in the posterior part of vasomotor center, which lies in **nucleus of tractus solitarius** in medulla and pons.

Function

Sensory area receives sensory impulse via glossopharyngeal nerve and vagus nerve from periphery, particularly, from the baroreceptors. In turn, this area controls the vasoconstrictor and vasodilator areas.

MOTOR (EFFERENT) NERVE FIBERS TO HEART

Heart receives efferent nerves from both the divisions of autonomic nervous system. Parasympathetic fibers arise from the medulla oblongata and pass through vagus nerve. Sympathetic fibers arise from upper thoracic (T1 to T4) segments of spinal cord (Fig. 101.1).

PARASYMPATHETIC NERVE FIBERS

Parasympathetic nerve fibers are the cardioinhibitory nerve fibers. These nerve fibers reach the heart through the cardiac branch of vagus nerve.

Origin

Parasympathetic nerve fibers supplying heart arise from the **dorsal nucleus of vagus.** This nucleus is situated in the floor of fourth ventricle in medulla oblongata and is in close contact with vasodilator area.

Distribution

Preganglionic parasympathetic nerve fibers from dorsal nucleus of vagus reach the heart by passing through the main trunk of vagus and **cardiac branch of vagus.** After reaching the heart, preganglionic fibers terminate on postganglionic neurons. Postganglionic fibers from these neurons innervate heart muscle.

Most of the fibers from right vagus terminate in sinoatrial (SA) node. Remaining fibers supply the atrial muscles and atrioventricular (AV) node. Most of the fibers from left vagus supply AV node and some fibers supply the atrial muscle and SA node.

Ventricles do not receive the vagus nerve supply. Few fibers are located in the bases of ventricles, but the functions of these nerve fibers are not known.

Function

Vagus nerve is cardioinhibitory in function and carries inhibitory impulses from vasodilator area to the heart.

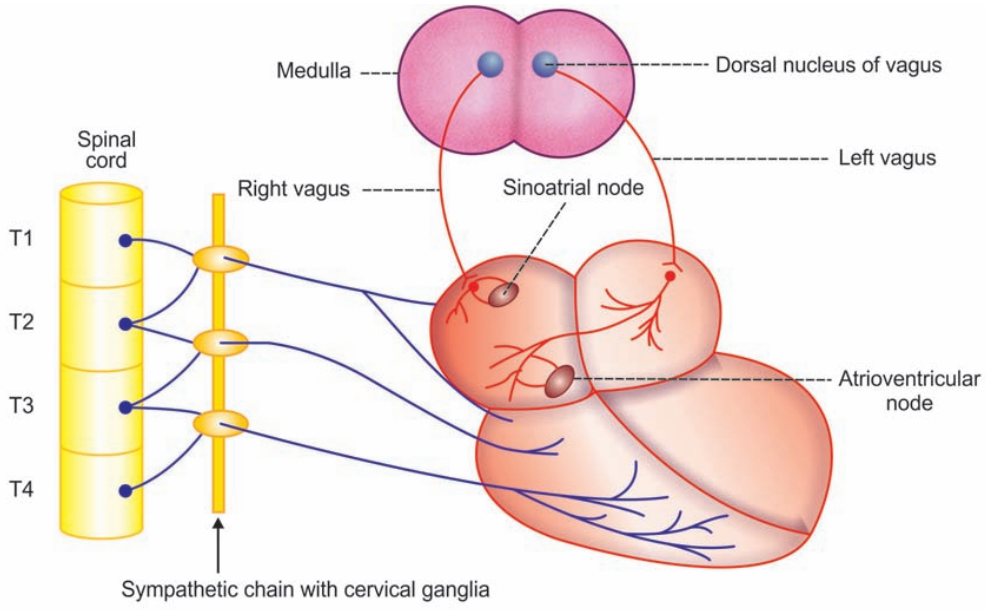


FIGURE 101.1: Nerve supply to heart

Vagal Tone

Vagal tone is the continuous stream of inhibitory impulses from vasodilator area to heart via vagus nerve. Heart rate is kept under control because of vagal tone.

These impulses reach the heart and exert inhibitory effect on heart. Heart rate is inversely proportional to vagal tone. In experimental animals (dog), removal of vagal input (by sectioning vagus) increases the heart rate. This proves the existence of vagal tone. Under resting conditions, vagal tone dominates sympathetic tone (see below).

Impulses from different parts of the body regulate the heart rate through vasomotor center, by altering the vagal tone. Vagal tone is also called **cardioinhibitory tone** or **parasympathetic tone.**

Effect of Stimulation of Vagus Nerve

Effect of stimulation of right vagus nerve – Vagal escape

Right vagus supplies mainly SA node. Stimulation of right vagus in experimental animals such as dog, with a weak stimulus causes reduction in heart rate and force of contraction. Stimulation with strong stimulus causes stoppage of heart due to inhibition of SA node. If the stimulus is continued for some time, the ventricle starts beating; but the rate of contraction is slower than before. This is because of vagal escape.

Vagal escape refers to escape of ventricle from inhibitory effect of vagal stimulation. If stimulation of vagus nerve is stopped, heart starts beating normally (Fig. 101.2).

Cause for vagal escape

Stimulation of right vagus stops the heartbeat due to inhibition of SA node and atria. However, ventricles are not supplied by vagus. So, the ventricles are not inhibited by vagal stimulation. Because of this, when stoppage of heart beat is continued for some time (by vagal stimulation), a part of ventricular musculature becomes pacemaker and starts producing impulses. It results in contraction of ventricles, which is called vagal escape.

Thus, vagal escape includes only ventricular contractions. However, the rhythmicity of ventricular muscle is less and it is about 20/minute.

Effect of stimulation of left vagus nerve – heart block

Left vagus supplies mainly the AV node. Stimulation of left vagus in dog with a weak stimulus causes a slight

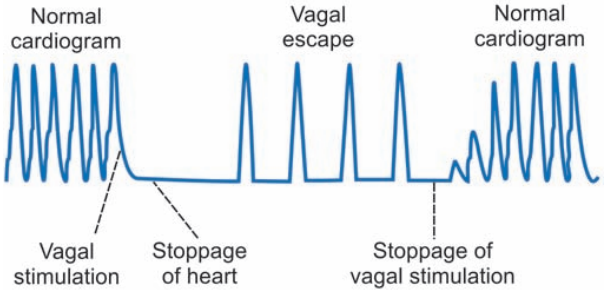


FIGURE 101.2: Effect of vagal stimulation on frog heart

reduction in rate of ventricular contraction. Stimulation of left vagus causes inhibition of AV node. Because of inhibition of AV node, some of the impulses from SA node are not conducted to ventricles. This is called the **partial heart block.** The ratio between atrial contraction and ventricular contraction is 2 : 1, 3 : 1 or 4 : 1, depending upon the strength of stimulus.

Stimulation of left vagus with strong stimulus causes stoppage of ventricular contraction, which is called **complete heart block.** This is because of the complete inhibition of AV node. The prolongation of stimulation causes **idioventricular rhythm,** which is different from the rhythm of atrial contraction.

Mode of Action of Vagus Nerve

Vagus nerve inhibits the heart by secreting the neurotransmitter substance known as **acetylcholine.**

■ SYMPATHETIC NERVE FIBERS

Sympathetic nerve fibers supplying the heart have cardioacceleratory function.

Origin

Preganglionic fibers of the sympathetic nerves to heart arise from lateral gray horns of the first 4 thoracic (T1 to T4) segments of the spinal cord. These segments of the spinal cord receive fibers from vasoconstrictor area of vasomotor center.

Course and Distribution

Preganglionic fibers reach the superior, middle and inferior **cervical sympathetic ganglia** situated in the sympathetic chain. Inferior cervical sympathetic ganglion fuses with first thoracic sympathetic ganglion, forming **stellate ganglion.** From these ganglia, the postganglionic fibers arise.